THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—45TH YEAR SYDNEY, SATURDAY, JANUARY 18, 1958 No. 3

Table of Contents.

[The Whole of the Literary Matter in THE MEDICAL JOURNAL OF AUSTRALIA is Copyright.]

	Page
ORIGINAL ARTICLES—	
The Sir Richard Stawell Oration—Five Men, by Sir Daryl Lindsay	61
Thyroidectomy: One Thousand Cases. I, by Alan Pryde	66
Subdural Hygroma, by J. Bryant Curtis	69
Atopic Dermatitis (Generalized Neurodermatitis): The Atopic Dermatitis-Asthma-Hay Fever Syndrome, by Emil Milder	72
Serum Glutamic Oxalacetic-Transaminase Activity in the Diagnosis of Myocardial Infarction and Hepatic Necrosis, by M. N. I. Walters and Patricia G. Littlejohn	76
REVIEWS—	
Modern Medicine for Nurses	80
Kinetics and Thermodynamics in Biochemistry	80
Dermatologic Formulary	80
BOOKS RECEIVED	80
LEADING ARTICLES—	
The Murray Report	81
CURRENT COMMENT—	
Primary Lymphœdema	82
Fluoride Dentifrice and Stomatitis	82
The Surgery of Portal Obstruction	82
Abstracts of Soviet Medicine	83
ABSTRACTS FROM MEDICAL LITERATURE—	
Pædiatrics	84
Orthopædic Surgery	85
SPECIAL ARTICLE—	
Rehabilitation: I. The Role of the Family Doctor	86
BRITISH MEDICAL ASSOCIATION—	
Victorian Branch: Scientific	87
OUT OF THE PAST	87

	Page
CORRESPONDENCE—	
Kala-Azar in a Child of Five Years	88
"Be Strong and of a Good Courage"	88
Carcinoma of the Lung: Always a Suspect	89
Plants, Including Fungi, Poisonous or Otherwise Injurious to Man in Australia, Series V	89
Sprue	89
Acute Appendicitis in a Centenarian	90
Immunization of Rh-Negative Post-Menopausal Women by Rh-Positive Blood Transfusions	90
POST-GRADUATE WORK—	
The Post-Graduate Committee in Medicine in the *University of Sydney*	90
Federation of Country Local Associations (New South Wales)	91
Clinico-Pathological Conferences at Sydney Hospital	91
DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA	91
THE COLLEGE OF GENERAL PRACTITIONERS—	
Proposed South Australia Faculty	92
CONGRESSES—	
Fourth International Goitre Conference	92
NAVAL, MILITARY AND AIR FORCE—	
Appointments	92
NOMINATIONS AND ELECTIONS	92
DEATHS	92
DIARY FOR THE MONTH	92
MEDICAL APPOINTMENTS: IMPORTANT NOTICE	92
EDITORIAL NOTICES	92

The Sir Richard Stawell Oration.[1]

FIVE MEN.

By SIR DARYL LINDSAY, LL.D.
(*Honoris Causa, Australian National University, Canberra.*)

I HAVE chosen the title of "Five Men" for my talk this evening for the reason that their names are of your profession, and I thought perhaps that a layman's observations on them might be of some interest to you. Four of them are great men who have left an indelible mark in their achievement, and their names will go down in history. Sir Henry Newland is fortunately still with us. The fifth man was not a great man, but he was an exceptionally interesting one, and it is because of my intimate knowledge of him that I feel justified in bringing him into so holy a circle.

It is not for me to assess the standing of these men in your profession—that is outside my knowledge; all I can attempt to do is to try to give you a picture of them as I see them from my angle of vision. Most people show different sides of their character to different people. I will attempt to give you the side they showed to me. The first name on my list is Hamilton Russell.

[1] Delivered at a meeting of the Victorian Branch of the British Medical Association on October 2, 1957, at the Royal Australasian College of Surgeons, Melbourne.

Hamilton Russell.

Hamilton Russell as I saw him had a sensitive and aristocratic mind. I understand that he was a great and original surgeon, who influenced all who worked with him and under him. His name will always be a legend at the Alfred Hospital.

But outside his profession I think of him as a lonely man, who kept the door closed on his innermost thoughts. Although friendly, he held himself a little apart and seemed a mixture of wisdom, cold cynicism and on occasion great kindness. He had the face of an ascetic priest of a very high order, but was lacking in red blood and a real sense of humour; I never felt that he was a happy man.

I was about 15 or 16 when I first met Hamilton Russell at the dinner table of a friend of my father. He said very little, but I was fascinated by his aristocratic and almost saintly face, with its smooth, ivory-like skin.

I did not meet him again until after the first World War, when I got to know him more intimately. He used to ask me to dine with him at Cliveden, generally alone, which was a great compliment to a young man; but Hamilton Russell seemed to cultivate young men. Sometimes he would play to me for an hour—he had a most

sympathetic touch on the piano — or would tell me about Jan Kubelik and Paderewski and other great figures in the musical world. He was interested in art, and supported and helped some of the younger artists of the day; but young as I was, I did not feel that he had any particular taste in this direction.

With the arrogance of youth I would often differ with his opinions about art; but with a sublime indifference — born, I dare say, of great wisdom — he would go to sleep, later to wake and adroitly change the subject.

He had a habit of ignoring anything or any subject he did not wish to discuss, and the conversation was often interrupted by periods of disconcerting silence — or sleep. He reminded me of Wilson Steer, the great English painter, who had the same trick of going to sleep — or appearing to go to sleep — and, like Steer, when he woke he would make some very profound remark.

He seemed to me to be very preoccupied, and even a little curious, about people we both knew, and he was particularly interested in their domestic relations and had very fixed ideas as to the basis of domestic happiness. He once gave me an instance of what in his opinion was the perfect marriage. He did not know—and I could not tell him — that I had spent nights and nights in trying to patch up a dreadful domestic mess in that household. It seemed to me that his psychology was sometimes that of the wise virgins.

I saw a great deal of "Hammie" one way and another — at the Hugh Devines' or the Maclures' at dinner, and sometimes he would telephone and say he was coming down to tea that afternoon. He might arrive at 12.30 for lunch — rather devastating to my wife, who generally rose to the occasion. One day when we were sitting down to lunch the telephone rang. It was the Alfred Hospital — they had been waiting for him for an operation since 11 o'clock.

An absent-minded man, Hamilton Russell was the worst driver I ever sat beside. I am not a nervous person and know the thrills of steeplechasing, but he terrified me. He once drove through the intersection of Collins and Swanston Streets at 5.30 against the policeman, with screaming brakes and curses hurled at him from all sides; but he drove serenely on, oblivious, as far as I could see, that we had escaped a smash by inches. A six-day tour in England reduced me to a nervous wreck, and I invented a lying excuse to leave him at Oxford and return to London safely by train. I am glad that I did, since the next time I saw him in London he had with him a nephew with a broken collarbone—the result of "Hammie" getting into reverse gear going up a hill in Bath.

He once took me with him on a round of visits to leave Christmas presents for the children of some of his young medical friends. He gave a doll to a lovely little girl, and after he had patted her on the head we drove away. After one of those periods of silence he said: "Charming child — a pity she has to grow into a woman." It was an observation that made me think, and threw a certain light on his life and possibly on why he remained a bachelor.

I am pleased to think that I was partly responsible for the portrait of him that hangs in this College. Alan Newton and Fay Maclure asked my advice; I suggested George Lambert, and it was left to me to arrange a meeting between these two very different characters. This was not very easy to fix; Lambert was a brilliant, arrogant and conceited man, and not easy to nail down. In his opinion he was just as important in his profession as Russell was in his. He wrote: "What manner of man is your friend Russell? Is he the sort of man I would be interested to paint?" Lambert was in Sydney and demanded that Russell should come to him. "Hammie" was not used to being treated in so cavalier a manner, and it took all my diplomacy to get him up to the scratch; but I had warned Lambert that the old man had the habit of dropping off to sleep. Lambert wired back: "Tell your friend Russell to bring with him some kind of dope to keep himself awake, otherwise I shall paint him asleep — the idea rather intrigues me."

"Hammie" returned to Melbourne after the first sittings a somewhat chastened man, and asked me to lunch. "Extraordinary man, Lambert", he said. "Talked to me like a stable boy."

I found out later that if "Hammie" went to sleep Lambert would shout "Hold up there!" or crack a stock-whip. Lambert was a wonderful mimic and a great *raconteur* and had a charming baritone voice, so he kept Russell awake by singing and telling him stories. But if the head began to drop, out would come the stock-whip.

I believe Lambert spent over 60 hours on this portrait, and they ended up great friends with a vast respect for each other. The result is one of the best portraits painted in this country, and shows to perfection Hamilton Russell in his later years. It is a penetrating likeness of a great surgeon by one of our greatest painters.

Henry Simpson Newland.

The first time I saw Henry Newland was in France in 1916. He was then Surgical Specialist in the Third Australian Casualty Clearing Station, which was stationed near Poperinghe. He was pointed out to me with great pride by one of his theatre orderlies, an old friend of mine, now a very prominent business man in Melbourne. I learned from the orderly that the unit had just come back from the front, and that during the Bullecourt stunt the Colonel had taken all the major cases and operations for the first two days with practically no sleep at all. My memory of him then was of a spare, wiry-looking man with a serious face and lips drawn close together in concentration as he passed us on the duckboards. Little did I know that he would be my commanding officer two years later, when I was posted to the Queen's Hospital, Sidcup, as official medical artist.

My going to Sidcup came about through a casual meeting with Colonel Jock Anderson and Sir Neville Howse at Horseferry Road — they wanted somebody who could draw medical diagrams for Newland. Would I go down at once and see Newland? I was due back in France the next day, but they fixed up an extension of leave, and it was not until months later that I discovered I had been "A.W.L." for 30 days. Nobody seemed to know where I was.

I reported to the Colonel the next morning; he was on the way to the theatre, and he asked me to come along and we would have a talk at lunch-time. I can see him now, standing with his gloved hands clasped together waiting for the patient to come in from the anæsthetic room. He explained to me that he was going to do the second stage of a rhinoplasty for the restoration of the nose; he said I could leave if it upset me. I had seen a good deal of blood one way and another, and it did not worry me. He got to work explaining a point here and there, but mainly he was absorbed in the job.

I became fascinated by his dexterity, but I was really more concerned with my own thoughts: how was I going to translate what looked like a mess of flesh and blood into a diagram that a student could understand? We had a talk at lunch-time, and I confessed and told him that Jock Anderson and Sir Neville Howse had "sold him a pup" in me, that I knew nothing about anatomy, and it was doubtful whether I had the capacity to do the job. With a smile on his rather severe face, he asked me if I would try it. I have no regrets, as I became intensely interested in the work and grew to have the greatest respect for this highly imaginative surgeon.

I soon learned that the hospital staff was rather in awe of the Colonel. He was a disciplinarian and kept everybody on their toes. He never spared himself and expected everybody to keep up with him. It was not until I had worked under him for some months that I had the opportunity to "get under his skin". He took me to dinner in London one night, and over a bottle of claret told me something of his life. He told me a story about Shakespeare, another about Sir Arthur Keith, whom I was to know later, and talked of other things. I realized that

behind the blue-grey eyes was a man of wide knowledge and scholarship with a great understanding of humanity. We have shared many a bottle of wine since, and the conversation has always been to my advantage.

Outwardly a rather austere man until one got to know him, he kept his emotions under control — I believe there is a touch of shyness in his make-up. But I remember once when his controlled emotion got the better of him. It was the day of the armistice, 1918. The Colonel had been to the dispensary, and we met on the ramp and passed the time of day. "Interesting news, Lindsay", he said, and might have been commenting on a minor topic in *The Times*. But as he spoke he was unconsciously snapping some tubes of tablets in his hands, and the tablets were falling on the ramp. It was, I think, a deeply-felt suppressed emotion of a man who had seen the war in all its phases, and now it was over.

Few men in the medical profession in Australia have received more honours than Sir Henry Newland, and few have been more worthy of the honours which have been showered upon him. During the first World War he received the D.S.O.; in 1919, the C.B.E. In 1928 he was knighted for his great services to the profession. Then followed F.R.C.S. (Honorary), Edinburgh; LL.D. (*Honoris Causa*), University of Melbourne; D.Sc. (*Honoris Causa*), University of Western Australia; Gold Medal of the British Medical Association in Australia; Gold Medal of the British Medical Association (England).

This is a great record of achievement. But as I see him, he never worked for honours; they came his way naturally for his great service to his profession and to his country.

Apart from his ability as a great surgeon and the honours that have come his way through his profession, he has other and equally important qualities. Henry Simpson Newland has a solid background; he comes of a family of fine traditions, and one has only to look into the Newland family record in South Australia to know why he is the man he is. His father and grandfather were great pioneers, and their name is writ large among those early settlers who laid the foundations of that State. Sir Henry Newland has inherited this fine tradition of service to his country. Although not large in stature, in character and intellect he is an outsize among men. He is a man who has always put his ideals — and they are high ones — before office.

A man always ready to champion any good cause, he has not only played a leading part in the medical history of Australia, but has applied his imaginative and logical mind to civic affairs in the public interest. I feel that he has most, if not all, the qualities we look for in a public figure — devotion to his profession, the highest principles, a wide knowledge of affairs, a humanitarian outlook — and on top of it all he is a man of the world and a delightful companion, who loves a good story over a bottle of wine. What more could one ask?

It has been my privilege and good fortune to have worked under him in a minor capacity, and in the years between to know him intimately and be numbered among his numerous friends. I can never repay his kindliness and understanding to a young subaltern.

Henry Tonks.

Had I not gone to Sidcup to work under Newland I do not suppose I would have met Henry Tonks. Tonks had taken medicine as his profession, and at 25 was demonstrator of anatomy at the London Hospital and acted as house surgeon to Sir Frederick Treves. A year later he took his F.R.C.S. Treves thought highly of Tonks and predicted for him a brilliant future as a surgeon. During these years Tonks had been studying drawing at the Westminster School under Brown, and when Brown was offered the professorship at the Slade School of Art at the University of London he asked Tonks to join him as teacher of drawing and anatomy. Tonks, who knew his own mind, did not hesitate, and at 27 threw up a brilliant future in medicine to become an artist. Some years later he succeeded Brown as professor, and under his direction the Slade School became the most important art school in England.

My first meeting with Tonks was at Sidcup, where he was putting in a day a week doing diagrams and drawing for Harold Gillies. I was struggling with a drawing and became aware of being overlooked by a tall, hatchet-faced man, who looked like a cross between a Roman emperor and a wedge-tailed eagle. He asked me what I was doing, and I said "Trying to draw". He said "I'm glad you said 'trying', which is the best that can be said of it; but I think I may be able to help you".

Thanks to the kindness of Henry Newland, I was allowed to go to the Slade one day a week to study under Tonks. The Slade opened up a new world to me. Under Tonks it had produced the most brilliant painters and draughtsmen of the day — Philpot, McEvoy, Orpen, and many others; but the most outstanding of them all was Augustus John, whom Tonks described as the most gifted student he had ever had. John, a Welshman, was so full of energy and spirits that he could not keep still, and one day, to use Tonks's words, "In a moment of . . . exuberance took a high dive into the sea, hit his head against a rock, and came out a genius". Tonks, with his piercing hawk-like eye, was an intimidating person, and the students at the Slade were terrified of him. There were stories of girl students jumping out of second-story windows or wanting to drown themselves at a critical word from the master. To one, after looking at a dismal drawing for some minutes, he said "Have you ever done any washing?" and, being answered "yes", added "Don't you think you are wasting your time here?".

My third lesson from Tonks led me into saying "I'm sorry, but I don't know what you are talking about. I know the meaning of the words, but not their application to art." This so staggered Tonks that he stalked from the room and an hour later sent for me. The other students looked at me with sorrow as one condemned to the gallows. It was an interesting conversation — or argument — and ended in my being asked to supper with him the next Sunday night.

I did not realize until later that to be asked to supper at the Vale on a Sunday night was to be admitted into the holy of holies. On that night there were Wilson Steer, George Moore, Tonks and myself, and I was put through the hoops. There was no guessing and one had to stand up to one's opinions.

I had many dinners at the Vale, and I never heard better conversation anywhere. It was ribald and full of meat, with Tonks calling the tune. Underneath his austerity Tonks was a shy, modest man, but he had the most fastidious taste of anyone I have met. This applied to people, painting, conversation, writing, food and wine. He demanded the best and would not tolerate anything second-rate. The dinners at the Vale were perfect — never more than four, which Tonks held was the perfect number for good conversation.

The food was excellent — soup or fish, lamb or an Aylesbury duck, cheese and fruit, and the best wine that Berry could provide. Although he lived simply, it was always the very best for his friends.

Mary Hutchinson, the wife of the celebrated King's Counsel, described him as being a mixture of "a duke, a monk and as wise as a countryman with his plough". These qualities he showed to his friends; but his art, his great passion in life, he kept to himself. He was a proud man, and if he had a weakness it was that he could not tolerate criticism of his work from ill-informed people. I was present once when General Furse, Director-General of Medical Services, and an old friend of Tonks, called. We were looking at a pastel drawing that Tonks had done. It happened to be a drawing of my wife which, I think, is a masterpiece of draughtsmanship. Furse, with the arrogance of office, said "Surely the left eye is a little wrong, Tonks". Tonks, white in the face, said "You have looked at this drawing for three minutes. I have looked at it for five days. How dare you pass an opinion?".

Furse was shown the door and told not to come back until he could mend his ways.

There is a very good picture of Tonks in the "Lives of the Fellows of the Royal College of Surgeons":

> He was very tall. Very thin and very caustic, living the life of an ascetic epicure. But a few minutes conversation would disclose humour and a sympathy with youth. He nourished the most conscientious side of everybody he influenced and to know him was to be influenced at once. The spell of trying to gain or keep his approbation never broke. His face did not easily register pleasure but the brilliant eyes over the formidable nose glowed with sincerity and positive hunger to behold excellence.

Tonks was without exception the most interesting and remarkable man I ever met. He was a great student, widely read in the classics and the best modern writers. He had no time for half-baked intellectuals. He was a magnificent draughtsman, and possibly the finest teacher that England has produced in the last 100 years. He knew practically everybody who was worth knowing in his day, and his comments on people, from prime ministers down to servant girls, were profound and very much to the point. My friendship with Tonks, which began at Sidcup, lasted until a few days before his death. Joseph Hone, in his "Life" of Tonks, wrote that he opened his heart to two people, Collins Baker and Daryl Lindsay, and this, I feel, is the greatest tribute that has come my way. His friendship and advice changed the whole course of my life.

Fay Maclure.

It would be wrong to say that Fay Maclure was a typical Australian. He was much too rare a person for that broad term; but no one could ever mistake him for anything else but an Australian, as there was something in his make-up that was racy of the soil of this country. With all his brilliance as a great surgeon, he still belonged to the type of Australian that leaned against veranda posts with his hands in his pockets and his hat tipped over his eyes.

I first met him at Sidcup, and the friendship lasted until I saw him a few days before his death. At Sidcup he was known as "Peter Doody" because of his rather unmilitary appearance — an ill-fitting service jacket, puttees badly put on and possibly inside-out, a battered cap, and a habit of drooping his mouth when thinking. His dry sense of humour made him popular with everyone.

It seems scarcely fitting that a layman should comment on the technicalities of surgery; but in the year when I was at Sidcup, and since, I have seen many eminent surgeons operate, and this experience has given me the basis on which, with all humility, I feel I can form some kind of opinion. Technical skill must be one of the first attributes of a great surgeon, but it is by no means the end-all of the business. Unless it is allied to an analytical and philosophical mind, it can become just a piece of highly skilled carpentry.

To watch Maclure make an incision was to realize that here was a man who knew exactly what he was going to do and why. (He once said to me that in teaching students "why" was the most valuable word in the English language.) There was no indecision — a knife directed by a mind that was clear and full of confidence — and so it went on through all the stages of an operation.

But to me there was another side of his work that interested me even more — his originality of thought and his acute observations; every case was a new problem, and he brought to it something essentially his own. I believe his colleagues and the students who came in daily contact with him in the consulting room and in the lecture theatre would agree that he had the qualities of a highly skilled surgeon combined with the profound knowledge of a great physician. He once said "To operate is the last resource of the surgeon".

If Fay Maclure had not turned his mind to medicine, he would have had just as brilliant a career in science or in the higher branches of the Criminal Investigation Department. Quite recently Dr. Murray Blair told me a story which illustrates to perfection his powers of observation.

> A young man was brought into the out-patient department at the Alfred Hospital, limping on his right leg. "What's the matter with this bloke?", asked Maclure. There were various guesses, from housemaid's knee to tuberculous hip, high blood pressure, etc. "Don't guess", said Maclure, "didn't you notice his watch chain? Five medals for football and athletics. What is the most common leg injury of a footballer? Internal derangement of the knee joint. Now go and examine him." It is hardly necessary to add that he was right—the deduction was worthy of Sherlock Holmes.

Born and bred in the country, he had an intimate knowledge of the land and an understanding of those people who live close to it. To these things he brought the same observation and balanced judgement as he brought to his profession.

I was fortunate in accompanying him on two trips to the back country of New South Wales. Everybody— squatters, country practitioners, publicans, boundary riders and deadbeats—knew or had heard of the "Doctor" and by bush telegraph knew of his "coming through". Each had his tale to tell; and as time is of little account in such places, we would sit in the shade of a tree or lean against a fence and hear the local news. I see him now, sitting in a dark hut at the bedside of an old boundary rider over eighty, who was in a pretty bad way. The old man rambled on — about the early days when men were men and the horses worth throwing a leg across. And the woman who had recently lost her husband, cheerfully battling on with 2000 sheep in rough country, her only help a daughter — and they did their mustering on bicycles. Maclure had an eye for character, and there was always a poker-faced wisecrack or a sympathetic word for these simple people living the hard way; but mostly he listened and occasionally went to sleep. He had a capacity for being able to sleep anywhere, at any time, and in any position. I once saw him, while talking to a tank-sinker on a blistering day, with the temperature about 110° F., lean back over the radiator of his car and go sound asleep. These are among the things I like to remember him by.

Fay Maclure seemed to me to be one of those rare people born knowing most of the answers. He had a quiet sense of humour, a deep understanding of humanity and an amazing fund of common sense. These things he brought to the practice of his profession. His philosophy of life and his high ideals are perfectly expressed in the words he used in the unveiling of the Memorial Tablet at the Alfred Hospital on September 28, 1948. What he said that day is a lasting memorial to himself and an inspiration to all who aspire to follow the standards set by Hamilton Russell and himself.

Robert Charles Lindsay.

The last man on my list is my father. He was educated in Belfast and Glasgow, where he took his M.D. degree. He came to Victoria in the early 1860's and put his plate up at Creswick, then a thriving mining town of some 20,000 miners, mostly living under canvas. He occupied one of the few weatherboard houses, and there is an amusing photograph of him standing in front of it in a frock coat and very high top hat.

A few years later he married Jane Williams, the daughter of Thomas Williams, the Fijian missionary. My mother was the first white girl to be born in the islands. She was 19 years of age and had just left boarding school when she married my father, and during the next 20 years they were very busy producing 10 children.

As I am the second youngest of 10, my memory of my father was from middle age until he died at the age of 76. He was a rather stocky, well-built man who carried himself very straight and marched rather than walked. He was something of a dandy, dressing in morning coats, tweeds and a strange assortment of hats — square-topped bowlers of different colours and, in summer, panamas with flowing

pugarees. He wore a flower in his coat, and always smelt of lavender water and chloroform.

As a small boy I got to know him very well. I say this for it is not given to every son to know his father intimately. In his latter years, as I remember him best, he was a very charming old man — if you happen to like old men; I did. He drove good horses and was something of a figure in the district. Those were the days when men were judged by their horseflesh, and went to funerals not so much out of respect to the dead as for the joy of racing each other home or to the nearest hotel.

> I remember once coming back from a funeral with my father when we were passed by the local blacksmith, who had a bit of blood between the shafts. My father, I imagine, torn between the dignity of his profession and the love of the chase, said to the groom: "I think we might push along a bit, Ben." The hint was all that Ben wanted, but he left his run too late. The blacksmith came home an easy winner. My father bought the blacksmith's horse the next week.

He often took me with him on his rounds to hold the horses, and on many a night I sat outside lonely farm houses watching a restive horse. On the way home I would drive, and sometimes he would sing Irish songs—"Barney O'Hay" or "The Mountains of Mourne". He always held that the Northern Irish and the Scots spoke the purest English, and when in the mood would recount stories of the early days in short, crisp sentences. One night, passing a farm house, he said: "Remember that place — called out in the middle of the night — took a bullet out of a man's shoulder — I think one of the Kelly gang — not my business to tell the police."

On another occasion, in the Dead Horse Ranges, he pointed out a place where he was "stuck up" at 2 o'clock in the morning by a man who stepped out into the track and asked him the time. "Not much time to think — gold repeater down my boots — spurs into my horse and galloped into the bush. Fired two shots after me."

I believe he had a good practice. He invested what money he had in gold mines — always the wrong ones. He had many stories to tell of the mining field — the Midas, the Great Berry Lead Mines, and of the Australasia disaster, when they struck old workings and the mine was flooded and 22 miners lost their lives. He was one of the rescue party and described the incident as "feeling my way along a drive — up to the neck in rushing water — struck in the face by the hob-nailed boots of a floating miner — most unpleasant".

I think my father could be described as a well-educated man of his day. He read the classics, admired Disraeli and Lord Rosebery, and disliked Gladstone. He liked the pleasures of the table and good conversation, and would bring a guest home for dinner and call for a bottle of champagne when there was no champagne in the house, he suffering under the delusion that there was always a well-stocked cellar.

On his periodical trips to Melbourne he would come back flushed in the face with good living and stories of Fitzgerald, O'Hara and other medical men, and laden with exotic foods from Fletcher Chester's — tinned grouse, smoked salmon and limburger cheeses. As my brother Reg said, "The old man likes food that stinks".

Apart from a flair for clothes and literature, he had little æsthetic taste. He took a great delight in going to sales, and would come home with a buggy full of atrocities—tortured cane furniture, pictures of roses and swans painted on glass in red plush frames, and all kinds of other things. These he would smuggle into the house, to be found later by a critical family and held up to scorn.

He had a passion for cold baths, and on the coldest day would stand under the shower and count up to 500 in a loud voice so that everybody could hear. He always embarrassed timid guests by coming to the breakfast table pink in the face and rubbing his hands together — "Hope you enjoyed your shower this morning."

I believe my father was a good physician. The local chemists told me he wrote the best prescriptions they dispensed. How good a surgeon he was I do not know. He told my brother Lionel that he never minded operating except on the brain or for prostate. I have grave doubts about his surgery, as once as a boy I was called into his surgery to hold the hand of a shivering small boy who had a fishhook in the ball of his thumb. My father anæsthetized the part and proceeded to make an incision when I interrupted him and said: "Not that way, Dad, cut it from the top and it will come out easier." I was kicked out of the surgery. Subsequent experience suggests to me that I was right. My father had a great charm of manner and could get away with all sorts of things. I once remember him, with a wave of the hand and a few words to the guard, holding up the Adelaide express for five minutes while he finished a story to a man he was seeing off. He was fond of the sea and when bored by family life would go off on a cruise as a ship's doctor. He had an inquiring mind and was always poking his nose into strange places. At Hong-Kong he got himself into an asylum with a local doctor and arrived back at the ship to be greeted by screaming sirens and an irate captain at the gangway, who bawled to him: "Do you realize, Doctor, you have kept this ship waiting 10 minutes?" The old boy replied: "My dear fellow, what is 10 minutes?", and I dare say within half an hour was entertaining the captain over a whisky and soda.

He was, I believe, rather proud of his large family; but having helped to bring them into the world he took no further responsibility and left them to their own devices. He was utterly bored with domestic affairs, which he left to my mother. It was a case of "Janie, you deal with this", and he would stalk from the house, singing to himself. Janie generally did.

He was very popular and beloved of his patients, and to this day there are people still living in Creswick who refer to him as "the old doctor". My father was the product of an age before intense specialization — one of many hundreds — when the general practitioner had to turn his hand to anything, and often at a moment's notice. These men, modest in their way, with their sound common sense, in the aggregate made a great contribution to your profession, and I cannot help feeling a little proud that my father was one of them. I can only add that whatever his good points and his failings were, he was a great character.

I had intended adding a sixth name to my list. Although the bearer was not of your profession, she might easily have been — my mother. She had, I think, the qualities that would go to the making of a first-class physician; as it was, she was something of a junior partner to my father. She vaccinated all the children in the district, did all the minor jobs that bored him — lancing carbuncles and I believe on occasions setting bones — and was at the beck and call of anybody for moral and medical advice. The female population preferred my father and the male population my mother. She could cope with anything, from stopping a bank manager from blowing out his brains in my father's surgery to harnessing up a pair of horses in the middle of the night to go out to a mining disaster. How she managed to bring up a family of ten and put up with the irresponsibilities of my father is beyond me; she was a most remarkable woman.

Conclusion.

As I said when I set out to speak to you tonight, most of my greatest friends belong to your profession. They have, each in his way, added something to my knowledge of life. They have given me an understanding of certain things that I would not have known without their intimate friendship. If I have in any small way added something to their lives, I am very happy; but I wish to pay my tribute to a great profession—possibly the greatest of all professions — which has given me so much.

THYROIDECTOMY: ONE THOUSAND CASES. I.

By ALAN PEYDE,
Honorary Consulting Surgeon to the Thyroid Clinic, Launceston General Hospital, Tasmania.

Criticism: A Story of Mistakes.

THE old saying "one learns by one's mistakes" is not a happy one when applied to the surgeon, and still less so when applied to his patients. Realizing that I cannot add anything new to the vast and excellent literature about thyroid surgery, I thought a story of the mistakes I have made should be of some interest and possibly of some value.

A previous report covering 50 thyroid operations was published in THE MEDICAL JOURNAL OF AUSTRALIA of June 10, 1944. A second report covering 253 thyroid operations under local anæsthesia was read at a meeting of the Royal Australasian College of Surgeons, Tasmanian Branch, on May 22, 1948. This present series includes the latter number, and consists of 1000 consecutive thyroidectomies, of which I have performed 651 at the Launceston General Hospital and 349 in private practice, from August 12, 1943, to January 24, 1955.

I will discuss the mistakes from without inwards, as the operation would progress.

Incision.

The resultant scar is really of vital importance—primarily, I should say, because an ugly scar acts as a very definite deterrent to the patient's friends, who may therefore keep on postponing an operation that may become more and more difficult and dangerous. I have made some crooked incisions and some irregular ones. The method I now use is, with the patient's head flexed, to mark the central line of the neck with dye and to make two lateral dots. This gives the level and length of the incision. A scratch with a knife leaves a permanent scar. Probably the best method would be to mark out the line of the incision on the day before the operation, with the patient sitting up. I still get some surprises at the post-operative level of the scar.

So, though we all know that it is what lies beneath the scar that is the important surgical principle, I would stress the very great value of a barely visible scar in a visible area.

The length of the incision can vary with the size of the goitre, as also the level. If there is a natural skin crease, that can be utilized.

Closing the incision is important. The pretracheal muscles and fasciæ are sewn together with interrupted sutures, the lowest one taking all available tissue. I call this the "cosmetic suture", as it makes a thick barrier and prevents the skin from becoming attached to the trachea.

The drain tube, which I always use, is brought out laterally through the muscle and then through the wound close to one end. It is removed at 48 hours and leaves no scar.

Subcutaneous fat suture is unnecessary. Careful approximation of the skin edges with clips applied not very tightly seems the most satisfactory method.

Injection of Local Anæsthetic Fluid into a Blood Vessel.

Five cubic centimetres of 1% "Novocain" solution injected into a blood vessel can produce convulsions and unconsciousness. This occurred in one case when I was using local anæsthesia only. The patient, a female, aged 45 years and weighing 148 pounds, had a convulsive attack followed by unconsciousness lasting some two minutes. She had no recollection of it and the operation proceeded. The injection was presumably given into a vessel above the upper lobe of the thyroid. The precaution of aspirating before injecting must have been omitted.

After this, Dr. R. Y. Mathew, of the Commonwealth Health Laboratory, made some tests with rabbits. We found that four cubic centimetres of 1% procaine solution induced convulsions from which the rabbit recovered. Injection of eight cubic centimetres induced convulsions and death. So there appeared to be a fairly safe margin in the human. Now I always take the precaution of aspirating after five cubic centimetres have been injected before injecting further solution.

Incision of the Trachea.

On two occasions, including once in this series, I have accidentally opened the trachea. As a matter of fact it is very easily done. With the head extended over a pillow, the trachea presents as a convexity. Dissecting the gland from the surface of the trachea, especially if a knife is being used, becomes a very dangerous procedure. Less extension of the head and the use of scissors make for safety.

On the two occasions referred to, I immediately closed the wound in the trachea and proceeded with the operation. There was no complication.

Injury to Recurrent Laryngeal Nerve.

Paralysis of one vocal cord as an immediate post-operative result occurred on three occasions. On two occasions paralysis of one cord occurred some months after operation. This must have been the result of fibrous tissue formation in the region of the nerve. This rather makes me doubt the wisdom of routine exposure of the nerve. So I do not now look for the nerve, and when tying the inferior thyroid artery make the dissection as far as possible from the gland, retracting laterally the common carotid artery and exposing the inferior thyroid artery three-quarters of an inch from the gland.

Forceful pulling of the gland medially with pressure of a retractor on the deep lateral aspect of the gland could cause damage to the nerve; this was, I think, the reason of its occurrence in two of the cases mentioned. The paralysis has persisted, but speech appears normal.

Horner's Syndrome.

Interference with the sympathetic chain producing Horner's syndrome occurred four times. The drooping upper eyelid and contracted pupil were noticed at the first post-operative visit within a few hours. In one case the signs commenced to clear up within a week. In the others they took some months, and in one were present 12 months later. In this case I think I tied the sympathetic chain in mistake for the inferior thyroid artery. The latter is a very thin-walled vessel and goes into spasm with very little handling. I have made it a rule not to tie anything that is not pulsating.

Spasm of the Vocal Cords.

I have had spasm of the vocal cords occur on several occasions. It occurs at any stage of the operation and can be rather frightening. In all cases the spasm passed over and the operation proceeded without further trouble.

The introduction of muscle relaxants has been a very great boon, and their use has relieved the spasm almost immediately in two recent cases. I have had one case of spasm of the vocal cords 24 hours after operation. This was associated with some degree of post-operative hæmorrhage. Tracheotomy was required. This was prior to the introduction of muscle relaxants.

Pneumothorax.

Pneumothorax occurred twice. It was my practice to inject the local anæsthetic solution into the region of the upper and lower poles of the gland. On one occasion, with the needle in position at a lower pole, suction produced air. Post-operative X-ray examination of the chest revealed a pneumothorax. I have discarded the lower pole injection since then. It was a risky procedure, I realize now. I had used it in over 200 cases previously. Actually, not infrequently the apex of the lung can be seen bulging into the wound when the patient strains.

Tetany.

Post-operative tetany occurred 14 times. It may be more correct to say that it was observed. I believe it may be masked and not observed if it occurs late, say some weeks, after operation. The commonly known signs may be absent. This occurred once in this series to my knowledge, but there may have been others.

In the observed cases tetany was noted usually within 36 hours of operation, and all patients responded well to calcium therapy.

I do not think that tying both inferior thyroid arteries is a predisposing factor in the occurrence of tetany.

Post-Operative Hæmorrhage.

The wound was reopened in 10 cases because of bleeding deep in the wound. I always leave a small drain tube in the angle of the wound. It serves to drain a small quantity of blood, but may become blocked. So absence of external bleeding is no indication of absent internal bleeding.

The three cardinal signs of bleeding deep in the wound are increasing swelling, increasing tenderness and increasing discomfort, the last expressed by the patient in terms of feeling of tightness and difficulty in swallowing and in breathing.

I think it is better to reopen the wound early, rather than to delay, hoping that it will not be necessary.

In most cases the bleeding has come from the remnant of the thyroid gland. Rarely is a bleeding point found. The wound is packed, the ends of the gauze strips being brought out through a gap between the skin clips, which close the wound again. The strips and clips are removed in 48 hours. The resultant scar is almost as good as the usual one.

Tracheotomy.

Tracheotomy became necessary on two occasions. In each case bleeding within the wound had been diagnosed, but reopening the wound had been delayed. On each occasion an extremely rapid exposure of the trachea was performed with the patient in the bed and without any preparation, as the patient was now choking. Tracheotomy relieved the situation, and the patients were taken to the operating theatre for control of bleeding with packing, etc. Both recovered without further complication.

Serous Collection.

After removal of a large gland, particularly if substernal, a large hole is left. This does close up considerably, almost immediately as the lung expands, but a large raw area is left. It is occasionally worth packing the hole for 48 hours, especially if there is a general slight oozing of blood.

From the fourth to seventh day, sometimes a fluctuant swelling becomes apparent. This can be aspirated quite painlessly with a large-bore needle through the almost healed incision. The procedure may require to be repeated. The scar is not affected.

Infection in the Wound.

The usual post-operative course is for the wound to be well healed and the patient discharged from hospital on the sixth day. Occasionally a cloudy serous collection has been released on the fourth or fifth day, and the patient's stay in hospital may be a further day or two. In two cases the collection was frankly purulent and was released by probing the incision line. Healing was delayed some days. If the patient's temperature is raised and the wound area is tender on about the fourth day, I think the use of penicillin is justified.

Exophthalmos.

In two cases of toxic goitre gross exophthalmos occurred some months after operation. These might be included in the story of errors. However, I feel sure that the exophthalmos would have occurred anyway, and the causation is still obscure.

Exophthalmos, when an early sign in toxic goitre, usually diminishes with the other signs after thyroidectomy.

Pyramidal Lobe Enlargement.

The pyramidal lobe of the thyroid gland is present in about one in 10 cases. If a portion is left at operation it will enlarge and the patient will later (perhaps 12 months) present with a lump high in the mid-line of the neck. It may be as large as a walnut. I have had this happen several times in the earlier cases. It is essential to remove the whole of this lobe. It is necessary to have firm retraction of the wound and follow the lobe steadily up to its apex, sometimes for as much as three inches.

Treatment of the Recurrent Nodule in the Pyramidal Lobe.

Many recurrent nodules in the pyramidal lobe respond to thyroid therapy and rapidly become smaller, but never entirely disappear. Those that do not respond may require excision if they are unsightly. This is a small operation, but not to be undertaken lightly. The nodule is closely attached to the trachea or cricoid cartilage and has a good blood supply. An incision directly over the nodule is the best approach and leaves a well-hidden scar.

Recurrence after Thyroidectomy.

On several occasions the portion of the thyroid gland left at operation has enlarged. I usually leave a piece measuring three by two by one centimetre on each side posteriorly. From six months onwards the patient returns with one or both lobes palpable or visibly enlarged.

It is essential at operation to remove all nodules, however small. In the case of an apparently solitary nodule it is necessary also to examine the other lobe, in which occasionally a small nodule will also be found. This procedure, and tying the inferior thyroid arteries, should reduce the incidence of recurrence.

The treatment of the recurrence is the same as of the pyramidal enlargement.

Crisis.

Crisis in thyrotoxicosis was a frequent cause of death before the introduction of the "antithyroid" drugs. Not uncommonly, death occurred before operation could be attempted. Particularly was this the case in nodular toxic goitre, in which the use of iodine was not nearly so effective as a pre-operative measure as it was in Graves's disease. The introduction of the thiourea type of drug revolutionized the treatment of thyrotoxicosis.

Post-operative crisis became uncommon and should now be rare. It is important, however, to remember the possibility of its occurrence if treatment with the drug is too short. It is wise before operating to continue with the drug until all signs of toxicity are gone. This was not realized in the early cases, and one death and several crises occurred as a result.

There were six cases of post-operative crisis in this series. Three of these occurred in the cases before the use of thiourea and in three cases after.

One patient died. The others recovered with the use of glucose in saline given intravenously, morphine and oxygen. Two were in a very precarious state for 36 hours.

The use of more modern drugs, such as chlorpromazine, may well be of very great help in the thyrotoxic crisis.

Death.

The death of his patient must be regarded as the surgeon's greatest mistake.

In this series there were seven deaths. It must be useful to study these in some detail.

CASE I.—The patient was a woman aged fifty years, suffering from Graves's disease. She had previously had cardiac failure, was grossly toxæmic, and had marked exophthalmos. Under "Avertin" and local anæsthesia, total

thyroidectomy was performed on June 8, 1944, and the patient was very well. On June 10, eighty hours after operation, she died from pulmonary œdema.

This case occurred just prior to our use of thiourea. The patient was a "bad risk"; but something had to be done, and it looked for a time as if we had been successful. The use of the antithyroid drugs before operation would have saved her life, I think. Today she would have been given radioactive iodine therapy, with almost certain cure and no operation.

CASE II.—The patient was a female, aged 33 years. She was four months pregnant and presented with a nodular goitre with marked signs of toxicity. She was treated with thiouracil for four weeks. Operation was performed on August 28, 1946, under local anæsthesia. The operation was uneventful and the patient spoke on return to the ward and took sips of fluids. She was found dead eleven hours after operation. Post-mortem examination revealed an embolus of the anterior cerebral artery.

Two glaring mistakes show here. The pre-operative preparation was too short. Now we do not operate until the toxicity is no longer present. Every patient to undergo thyroidectomy should have a special nurse for the first 24 hours. That, and the availability of a tracheotomy tray, are now routine requirements.

CASE III.—The patient was a female, aged 56 years. She had had a large nodular goitre for many years. There were no signs of toxicity. She had been easily short of breath on exertion for two months. Her blood pressure was 190/100 millimetres of mercury. Operation was performed on February 14, 1951. The anæsthetic consisted of the continuous intravenous administration of "Pentothal" and the local use of procaine. The large gland encircled the trachea. There was intermittent cyanosis throughout, in spite of a perfect airway. Four hours after operation she was distressed, with noisy respirations. Moist sounds were heard all over her chest. At 12 hours her temperature was 102° F., her pulse rate was 130 per minute, and her respirations numbered 26 per minute. Her temperature and pulse rate remained elevated, and she died 40 hours after operation.

The physicians regarded this case as a thyrotoxic crisis of unusual type. I have seen several typical post-operative crises in cases of apparently non-toxic goitre. They have occurred in middle-aged women with nodular goitres of long standing. Radioactive iodine investigations in a series of these have shown the patients to be euthyroid and not "minimal toxic", as they are regarded in some clinics. Nevertheless, I treat all such goitres as potentially toxic. Details may be seen under the heading "anæsthesia".

CASE IV.—The patient was a woman, aged 37 years, who presented with a nodular goitre which had been in evidence for 18 years. She had some difficulty in swallowing. There were no toxic signs. Her blood pressure was 170/105 millimetres of mercury. Operation was performed on June 13, 1951, under intravenous anæsthesia with "Pentothal" and local procaine. Half-way through the operation, laryngeal stridor developed. This was relieved, but she remained cyanosed. The vocal cords were normal and an endotracheal tube was passed and left in for three-quarters of an hour. Six hours after operation she stirred and opened her eyes. At seven hours she became cyanosed and her pulse weakened. Eight hours after operation she died. No post-mortem examination was obtained. The cause of death was given as cardiac failure.

This death opens up a lot of questions. Would this patient have died after hysterectomy or cholecystectomy? I do not think so. Why, then, did she die when she was not thyrotoxic? I think the vital period was the anoxæmia resulting from the laryngeal spasm. The fact that she had hypertension was also probably a factor.

CASE V.—The patient was a woman, aged 68 years, who had had a goitre for 15 years. Recently it had become more noticeable. She complained of occipital headache. On examination of the patient, in the mid-line of her neck there was a firm nodule, extending substernally. Her blood pressure was 190/90 millimetres of mercury. There were no signs of toxicity. Operation for removal of the nodule was performed on June 19, 1951, under intravenous "Pentothal" and local anæsthesia. Two further nodules were found, one deep on the right side and one deep on the left side, both substernal. Most of the gland was removed with some difficulty. Neither inferior thyroid artery was tied. Her post-operative progress was normal, and she was discharged from hospital with a healthy, healed wound on the eighth day. She reported back a month after operation, complaining of very bad headaches and of not sleeping. Her blood pressure was 185/90 millimetres of mercury. The neck appeared normal. Ten days later she appeared with gross swelling of the neck and some difficulty in breathing. The wound was opened and a great quantity of black, clotted blood was removed. The wound was packed and closed, and I thought the episode concluded. However, free oozing continued, and the wound was again opened and packed. At this stage the wound appeared as a black cavern, in which there was no discernible feature and no particular bleeding spot. In spite of firm packing, bleeding continued, and the wound was again opened and packed. Blood transfusions and all other aids were ineffective, and she became weaker and weaker and died six weeks after operation. No post-mortem examination was obtained. The cause of death was given as hæmorrhage.

CASE VI.—The patient was a man, aged 46 years, who was admitted to hospital with congestive cardiac failure and thyrotoxicosis on February 2, 1951. He was treated with antithyroid drugs, and was discharged from hospital on May 10. When examined on September 27 he was much improved. On November 18, 1951, he was admitted to hospital with acute appendicitis, and appendicectomy was performed. He progressed so well and appeared to be so well that it was suggested that he should have his goitre removed while he was still in hospital. On December 7 thyroidectomy was commenced under intravenous "Pentothal" and local anæsthesia (0·5% procaine solution). Half an hour after the commencement of the operation his heart ceased beating. The operation had proceeded quite normally until then.

Again the question arises, why did he die after successfully negotiating an appendicectomy? I think we must accept the fact that there is some risk in operating in the region of the deep tissues of the neck. Apropos of this, I think extravasation of blood in these same deep tissues causes distress out of all proportion to the quantity of blood. Nowadays this patient would have been treated with radioactive iodine and not surgically.

CASE VII.—The patient was a woman, aged 49 years. She had had a goitre for 25 years, and it was increasing in size. The goitre was nodular and extended substernally. X-ray examination showed that the trachea was pushed considerably to the right. There were no signs of toxicity. Operation was performed on July 18, 1952. Nitrous oxide, oxygen and cyclopropane anæsthesia was used, with local anæsthesia (0·5% procaine solution). Towards the end of the operation there were signs of cyanosis and collapse. A left pneumothorax was found and relieved. She never regained consciousness, and lived for 11 days. No post-mortem examination was obtained.

Comment.

The mortality rate for the 1000 cases was 0·7%. The cause of death in each of the seven cases was different. In only three were there signs of toxicity before operation. The other four had the following factors in common: (i) The patients were females. (ii) The patients were in the middle-age group. (iii) The goitres were nodular and of many years' duration. (iv) The goitres were moderately large, of an average weight of 200 grammes. (v) The goitres were situated deeply. (vi) The patients all had some pressure symptoms.

Anæsthesia.

I have acknowledged my mistakes in the injection of the local anæsthetic agent. Perhaps I may comment on the mistakes of the anæsthetists. On second thoughts, I will say only two things.

First, only a specialist anæsthetist should be employed for thyroidectomy. Secondly, I dislike the use of the endotracheal tube. It is unnecessary, however displaced or narrowed the trachea. When the tube is withdrawn the trachea may collapse.

I have seen this happen, and on this occasion stitched the wall of the trachea to nearby structures with success. On those rare occasions when a tube has been passed, I insist on its withdrawal before closing the wound.

The following is the method of anæsthesia I prefer.

Premedication consists of the administration of "Omnopon" (one-third of a grain) and scopolamine (one one-hundred-and-fiftieth of a grain), one hour before operation. For patients aged over 60 years, atropine (one one-hundredth of a grain) is substituted for the scopolamine.

The continuous intravenous administration of dextrose (4%) in one-fifth normal saline is commenced. Thiopentone is injected via the tubing. When the patient is asleep, the local anæsthetic agent is injected subcutaneously and deeper. The anæsthetic solution is 0·5% "Novocain" in normal saline, to six ounces of which is added one cubic centimetre of ephedrine sulphate solution, one in 1000. Thiopentone is injected via the tubing as required. Oxygen is administered intranasally as required.

I believe the continuous intravenous use of fluid a most important part of the operation. If the patient has been toxæmic, the administration of fluid is continued for a second pint, also if she has lost more blood than usual.

I have seen no ill effects whatever. Of the 349 cases in private practice, in 268 the anæsthesia was carried out in this manner. There was one death, and that one the death from hæmorrhage six weeks after operation. There were no complications of note as a result of the anæsthesia.

Conclusion.

The mistakes made during a series of 1000 thyroidectomies are discussed and some remedies for them suggested.

Acknowledgements.

I acknowledge with gratitude my debt to my associate physicians Dr. J. A. Newell and Dr. J. L. Grove; to the late Sir Alan Newton, who was most generous in his advice; to Dr. Henri Welti, of Paris, and Dr. J. E. Piercy, of London, for many practical demonstrations; and to Dr. Hugh Ramsay, anæsthetist. The operating theatre and nursing staff of the Launceston General Hospital and of St. Luke's Private Hospital eased tremendously the burden, particularly that of the post-operative care. To Matron N. Winwood and Sister E. King, of the latter hospital, I cannot be too grateful.

SUBDURAL HYGROMA.[1]

By J. BRYANT CURTIS,
Melbourne.

Of recent years there has been a great deal of interest in the subdural space in cases of head injury, but it has been concerned with cases of subdural hæmatoma complicating closed head injuries in the acute, subacute or chronic phases. The pattern of these has come to be fairly well understood, and most of us are ever aware of this possible complication, so that its possible existence is not often overlooked. On the other hand, it not infrequently happens that the finding of a chronic subdural hæmatoma is a most welcome surprise, when a less favourable condition has been suspected.

In general, it has been found that subacute and chronic hæmatomata have a good prognosis if treated adequately, and that acute subdural hæmorrhages have a very poor one, being usually associated with severe cerebral damage and laceration. In the same way, the condition of subdural hæmatoma occurring in newborn or young infants is also well recognized, largely owing to the work of Ingraham and Matson (1944).

The group of cases in which there occurs a subdural collection of clear, colourless or yellow fluid, usually following trauma, is not so well appreciated, and it is this group of cases which I intend to discuss. The condition has been variously described as traumatic serous meningitis and *pachymeningitis serosa*, but Dandy (1932) labelled it subdural hydroma, and by this name, or hygroma, it is most commonly known. Others have called it subdural effusion; but in the absence of certain knowledge of its origin, it is perhaps better to call it "subdural collection". From a study of the literature and from my own series of cases it would appear to be potentially rewarding if thought of, looked for and treated.

I have not been able to find much literature on the subject, the majority of papers being concerned with reports of very small series of cases. Dandy (1932) deals with it well. Payr (1916) described it as a complication of tangential gunshot wound injuries of the skull, and this old paper is most thorough and informative. It is interesting to find that Abbott, Due and Nosik (1943*a*, 1943*b*, 1943*c*, 1943*d*) described a similar complication occurring in 10 of 37 cases of brain injury secondary to blast concussion in the second World War. Naffziger (1924) drew attention to the condition and described the beneficial results of operative treatment, but gave no details of his cases. But it is McConnell in Dublin who has provided the greatest amount of information, with a series of papers discussing the clinical manifestations, X-ray and pneumoencephalographic findings and operative treatment (1941, 1942, 1943, 1944, 1951, 1953), and Wycis, of Philadelphia (1945), made an excellent contribution with a detailed report of seven cases.

The present paper presents a further series of 13 cases in adults and seven cases in babies, which I will discuss in separate groups.

THE ADULT SERIES.

The adult series divides into two clears groups—an acute group of six cases and a subacute and chronic group of seven cases.

Acute Group.

Of the six patients in the acute group, four died in the early stages, one died some months later, and one made a full recovery. All these cases were obviously patients with severe brain injury, being in a state of coma on their admission to hospital shortly after injury. Some showed signs of mid-brain injury. Operation was carried out in all cases because, after an initial improvement in the level of consciousness and neurological state, there was a progressive deterioration which was sometimes very rapid. Operation was carried out at periods varying from the first 24 hours to the fifth day.

A private patient had, in addition to his head injury, a traumatic tension pneumothorax and fractured right femur. On the second day, after a return of consciousness, there was a rapid onset of coma. Operation showed a subdural collection of clear fluid, under great pressure, on the right side. After evacuation of the fluid, the brain was slack and pulsating. However, the next day the diagnosis of traumatic fat embolism became clear with the appearance of widespread cutaneous petechiæ. Death occurred on the fifth day.

A patient, in the Royal Melbourne Hospital series, on admission was in a deep coma, with unequal pupils which were not reacting to light, and the plantar responses were extensor in type on both sides. The clinical picture suggested a mid-brain injury. Two days later surgical emphysema of the left side of the chest appeared. There was slight improvement over the next few days, although tracheotomy needed to be carried out. On the fifth day there was sudden deterioration in the patient's condition, and at operation one or two ounces of xanthochromic fluid was found in the right subdural space, under great pressure. Death occurred two days later, after temporary improvement.

Another patient, on admission to hospital, was in a deep coma immediately after a head injury. A subdural collection of blood-stained fluid on the left side was found after 36 hours. At autopsy, a large intracerebral hæmorrhage was found bursting on the inferior surface of the left frontal lobe.

In one of the five fatal cases in my private series the patient did not die for about six months. He was aged 60 years. On his admission to hospital he showed the signs of severe brain injury with deep coma, pinpoint pupils and extensor plantar responses on both sides. After an initial steady improvement there was a rapid deterioration on the third day. Operation revealed a large right subdural collection of clear fluid under pressure. After evacuation of the collection, the brain was slack and pulsating. There was a rapid improvement in the patient's conscious level, and he survived. But he was left with severe dementia and bilateral spasticity. Some six months later he died.

[1] Read at a meeting of the Victorian Branch of the British Medical Association on May 1, 1957.

One patient in the Prince Henry's Hospital series made a full recovery. He was aged 25 years and was admitted to hospital in a semi-comatose state, but responding. There were no localizing signs, but both plantar responses were equivocal. After some slight improvement, on the fourth day there was a rapid increase in his coma, right hemiparesis and spasticity of the right arm appeared and the plantar responses became extensor on both sides. At operation a large left subdural collection of clear fluid was released under greatly increased pressure. The surface of the brain was somewhat contused. The brain was slack and pulsating after it. On the right side no abnormality was found. There was an immediate improvement, with a slow return to full consciousness over seven to ten days and disappearance of the right hemiparesis. But about two weeks later it was seen that he had a fully developed type of Korsakov's syndrome, showing very poor memory, confabulation and euphoria and being fatuous. Over the next few months he made a full recovery, and has remained quite normal over the seven years in which I have examined him periodically.

Discussion.

On reviewing this group it is apparent that the results have been bad. But it is also clear that the subdural collection in the main has been associated either with evidence of severe brain injury, particularly in the midbrain, or with some other complication such as traumatic fat embolus. All patients presented with initial coma, and operation was carried out in all the cases because of the now classical indications of progressive deterioration in the level of consciousness, with or without the appearance of local neurological abnormalities. In all the cases the pre-operative diagnosis was made of intracranial hæmorrhage, presumably subdural or intracerebral, causing compression.

McConnell (1941) had eight cases corresponding to this group, of which four were fatal. The fatal cases were all associated with deep coma. None of the surviving patients showed initial deep coma; all improved eventually, two with lucid intervals, and all obviously had milder injuries generally.

Three of Wycis's cases were acute; none of the patients had initial signs of severe brain injury, and all recovered well.

Few of the other cases fall into this group.

Subacute and Chronic Group.

It is this group of seven cases which has received the most attention in the literature, and which has given the most encouraging results. Of this series, six patients have survived and one has died, and this death was probably preventible, as it appears to have been due to dehydration.

It may be of interest to recount my first experience with this type, which occurred only ten months ago. Although in the previous nine years I have seen none, since then there have been six further cases. I am convinced that this is because they have been specifically looked for, and this is the view of McConnell, Wycis, da Costa, Abbott, and others.

The first patient in my private series was admitted to hospital on July 5, 1956, shortly after a head injury. He was comatose, and showed signs of bilateral extensor rigidity with extensor plantar responses. The next day the coma was less and he was able to be roused somewhat; the extensor rigidity was less, but it was now observed that he had a left-sided hemiparesis. Over the subsequent few days there was a full recovery of consciousness, but the left hemiparesis persisted. On July 13 he showed confabulation and a mild Korsakov syndrome. By July 26 he had passed into a severe psychotic state, in which he called out in a loud voice every few seconds continuously day and night. The psychiatric prognosis was considered very poor, and it was decided to certify him as insane. Purely on the grounds that before taking this step I wanted to exclude definitely an organic lesion which could be treated, I operated on him on August 2. To my surprise, there was a massive collection of clear yellow fluid in the right subdural space, which was drained. Quite a tough, shaggy subdural membrane was present. No fluid was found in a left temporal burr-hole. Here the brain was under some tension, which was not excessive, and the arachnoid bulged into the dural opening. I will refer to this later.

After the right side had been drained, the brain was slack and pulsating, whereas previously it had not been. There was copious drainage for 48 hours. An immediate dramatic improvement occurred in his mental condition and the left hemiparesis decreased; but he was still very confused, confabulating and psychotic. The improvement was not maintained, and after two weeks he had reverted almost to his pre-operative condition, with the difference that he was not so noisy and the left hemiparesis was more severe than before.

Further operation was undertaken on August 18 through the old bi-temporal burr-holes, and there was a collection of fluid on both sides, which was evacuated. On this occasion bi-frontal and right parietal burr-holes were made, and large collections of the same yellow fluid were found over each frontal lobe, the left more than the right. They were all drained.

From then he made steady progressive improvement. Nine months later his mental state is good, although he is still preoccupied with himself. He is a medical statistician, and a colleague thinks that his intellectual capacity is as good as ever, although there are still memory and concentration defects. His left hemiparesis has almost entirely disappeared.

The second case (in the Prince Henry's Hospital series) was very similar. After a severe injury, recovery of consciousness was slow, but the patient then showed psychotic sexual symptoms and a well-marked Korsakov's type of syndrome. One month after the injury, when this state was unchanged, multiple bilateral burr-holes were made, and large bilateral collections were evacuated and drained. After two weeks he showed great improvement, and according to his relatives he had returned to his previous timid and rather shy state.

Since then the search has been extended more widely, and I shall refer to this again later. Burr-holes have been made in cases in which recovery, at first perhaps good, has later been retarded, particularly if there has been marked mental deterioration such as disorientation, confusion, confabulation or loss of memory. Five more cases have been found at operations which have been carried out from 18 days to 35 days after injury.

Two of these patients have made good recoveries. One, in the Royal Melbourne Hospital series, is improved, but confusion persists, and further operation is to be performed. One patient, in the Royal Melbourne Hospital series, was greatly improved from her stuporose state, but is still showing a marked Korsakov's syndrome. One died after operation from pulmonary complications.

Discussion.

The majority of cases reported in the literature are similar to this group. Dandy made little mention of the mental symptoms, but considered that this condition mimicked subdural hæmatoma. McConnell has not stressed the positive psychotic symptoms, but in this group has stressed the retardation of recovery, the confusion and the persistent dementia. Of six cases which he reported, one terminated fatally — that of a man, aged 54 years. In Wycis's series three correspond to this group, and all recovered well.

Normally it is considered (Penfield, 1924) that the subdural space contains no more than one or two cubic centimetres of fluid. If present, it is probably under the temporal lobes (Wycis, 1945). In these traumatic cases it is considered by most that the collections are the result of a tear in the arachnoid, which has a ball-valve effect, and that the fluid is cerebro-spinal fluid. The studies of Leary and Edwards (1933) indicate that the structure of neither the dura nor the arachnoid suggests any secretory properties or permeability. However, there are some difficulties in the way of accepting this simple mechanical explanation.

In one of McConnell's (1942) cases, a burr-hole made seven hours after the injury showed no fluid, whereas nine days later there was a large collection. But in one of da Costa and Adson's (1941) cases the tear was observed. However, it is difficult to explain the consistently high protein content found in the fluid, if it is cerebro-spinal fluid. In one of my series the protein content was 200 milligrammes per 100 cubic centimetres on the right side (the fluid was slightly yellow), and 2000 milligrammes

per 100 cubic centimetres on the left side, where the fluid was deep yellow. When it had been investigated by lumbar puncture, the cerebro-spinal fluid was normal in this chronic group.

It is probable that the explanation is a mixed one. When the fluid is clear and collects early, it is probably escaped cerebro-spinal fluid. If it collects late, it is probably a mixture of blood and cerebro-spinal fluid, which with breakdown has caused subsequent effusion. But that is only my personal view.

Clinical Picture.

The clinical picture in the acute group is fairly clear cut. The condition has been found at operation, which has been indicated by deterioration of the general level of consciousness of the patient, usually associated with the appearance or the accentuation of local neurological signs. The prognosis, which is bad in my series, appears to depend mainly on the severity of the initial brain injury. In the cases of milder initial injury the recovery rate has been good (Scott, 1942, 1943; Walsh and Shelden, 1937).

In the subacute group the outstanding feature has been the persistence of stupor and confusion or, with the improvement in the conscious level, the appearance of a psychosis. Focal neurological abnormalities have sometimes been present, but not consistently so. If present, they are relieved *pari passu* with the mental state.

But other groups have been described, apart from the blast injuries of Abbott *et alii*. Lanigan (1944) reported three cases following head injury, in which neither initial concussion nor amnesia was present at the time of the injury.

McConnell (1944) reported six cases in which the outstanding symptom was prolonged amnesia. McConnell has also reported a group of which I have no personal experience, but which appears to be most important and well worth further investigation. I refer to that group of cases of chronic illness following head injury which are labelled "post-traumatic syndrome" or "post-traumatic neurasthenic group". It is difficult to judge from his papers how his cases are picked; but he has reported (1953) 75 consecutive cases of "post-traumatic syndrome", in which 20 patients complained of headache only, seven complained of giddiness and 38 complained of giddiness and mental instability. He states that of these 65 had subdural collections. All had elevation of intracranial cerebro-spinal fluid pressure, although lumbar puncture pressure was normal. He is of the opinion that the fluid is cerebro-spinal fluid, and its presence is caused by an arachnoid tear which can be demonstrated radiographically. He states that characteristically this is shown by the subdural presence of air in the second 24 hours after a pneumoencephalographic examination (1953). He found this in 26 cases out of 34, and it was usually associated with a visible fluid level in the subdural air 24 hours after pneumoencephalography. I have no experience with this group or with these X-ray findings.

In view of these findings of McConnell and of similar ones of da Costa and Adson, it is curious that so little mention was made of the possibility of this condition when the post-traumatic syndrome was discussed by Trotter (1934, 1930). Symonds, Jefferson, Russell Brain and Cairns in 1942, during the war, also made no mention of it, although Cairns and Russell Brain made a brief mention of it in their paper in 1928, without going into any details of the clinical picture or the diagnosis. It appears that this group of cases needs more careful investigation before being labelled "post-traumatic neurasthenic state".

Operative Technique.

I have mentioned, in the first case of the subacute group, the failure to find a collection on the left side at the first exploration. But it was noted at the time that the arachnoid bulged through the dural opening and that the brain was tight. At the second operation, on the left side, it was only after the brain, with its arachnoid covering, had been greatly depressed away from the dura that the clear yellow subdural fluid flowed away copiously. In different directions the degree of coloration varied, as if loculation was present, with varying levels of protein in the loculations.

This is the essential point of the technique. Unless the fluid collection is massive and under the site of the dural opening, it may easily be missed.

Infant Series.

In the infant series of seven cases there are none of subdural hæmatoma in which either solid or fluid clots have been found. These are all cases in which subdural hæmatoma has been suspected after birth, injury or infection.

It is to Ingraham and Matson's paper in 1944 that we owe our increased awareness of the condition of subdural hæmatoma in infants, although there were occasional reports previous to this. But in the series of 98 cases which they reported then, there is a group of 10 which, I venture to suggest, cannot truly be classified as hæmatomata. These patients are described as having "excessive clear fluid with elevated total protein on subdural tap". I think this viewpoint is strengthened by Ingraham and Matson's statement that in only just over half their cases is there a definite history of trauma to the head, and that in almost half there was evidence of infection elsewhere in the body. Also, the presence of subdural collections complicating *Hæmophilus influenzæ* meningitis has been reported by McKay *et alii* (1950), and bacterial meningitis by Smith *et alii* (1951) and meningococcal meningitis by Steinberg and Murphy (1951), and these are further examples that trauma is itself not necessary for the presence of such collections.

These cases reported present a very real difficulty to the acceptance of the theory that the fluid present is cerebro-spinal fluid collecting as the result of an arachnoidal tear, and is in favour of the theory of an effusion into the space. As I mentioned earlier, probably both theories are right in different cases, traumatic or non-traumatic.

Of the seven cases reported here, there was a definite post-natal head injury in one case which occurred at the age of five months. Two days later vomiting commenced. Nine days after the injury subdural taps produced 25 cubic centimetres of xanthochromic fluid from the right side and none from the left. The following day a right burr-hole released more fluid. It may be said here that in none of these babies operated on has a membrane been found as described by Ingraham and Matson. Subsequent taps were performed on the right side, with the release of smaller amounts of fluid, and the baby was sent home, well, after 18 days.

In three of the cases there was a history of rapid or difficult labour. In two there was a story of probable infection, and in one case no relevant history was obtained.

One of the babies died, and the history was interesting. Pregnancy and delivery were normal, and the child was well for three weeks, when it refused feeds, became lethargic and had intermittent attacks of cyanosis. X-ray examination of the chest suggested a low-grade pulmonary infection. Later X-ray films showed heart enlargement. The head commenced to enlarge. Repeated subdural taps over 17 days produced a total of 64 cubic centimetres on the right side and 21 cubic centimetres on the left side, the fluid being deeply to mildly xanthrochromatic. Then at operation bi-temporal burr-holes were made and on both sides large collections of xanthochromatic fluid were found, which were drained. The child collapsed after operation from bronchial obstruction, and eventually died about one month later. Post-mortem examination showed that this baby had pyæmia with widespread small embolic abscesses.

The third type is illustrated in the following case.

One child (in my private series), aged sixteen months, had severe local symptoms in the left arm, a mild but definite hemiparesis and severe temper tantrums. There were dilated scalp vessels on the right side. Because of this an angioma was suspected, and a right carotid angiogram was

taken. This revealed a curved displacement to the right of the anterior cerebral artery, indicating a left frontal space-occupying lesion. Ventriculography confirmed this, and a left temporal burr-hole produced one to two ounces of clear fluid in the subdural space under increased pressure. After this the brain pulsated fully. Drainage was not employed, but there was escape of fluid through the incision for a further 24 hours. After operation there were no further fits, the temper tantrums disappeared and the left hemiparesis has almost entirely cleared up.

No ætiological factor could be found in the history of this child. The neurological findings are interesting, in that the baby had a hemiparesis on the same side as the lesion — a feature which is relatively common in chronic post-traumatic hæmatoma in adults, but rare in other space-occupying lesions.

Of these seven patients, six survived, and all have recovered very well. The one baby who died had a chronic pyæmic infection.

Summary.

1. Thirteen cases of subdural collections of fluid found in adults after head injuries have been discussed.

2. Six cases occurred in the acute phase, and of these five ended fatally and one in complete recovery. In the fatal cases there was evidence of severe or complicated brain injuries. The literature suggests that other acute cases follow milder head injuries, and that in these the prognosis is better if they are treated promptly by operation.

3. In seven cases the collection of fluid occurred in the subacute or chronic stage, and in these circumstances the prognosis appears to be much better. These cases have all been found in the last 10 months, and five in the last two months, when a careful search has been made for them. So, in my opinion, the total number is no indication of the frequency.

4. Seven cases occurring in babies have been found and treated. Trauma does not always appear to be the causative factor, and infection probably plays a definite role. The results in this group are good; six of the patients survived and recovered.

Acknowledgements.

Acknowledgement is made to the honorary medical staffs of the Royal Melbourne Hospital, Prince Henry's Hospital, Queen Victoria Hospital and Footscray Hospital for permission to publish the cases in which I operated.

References.

Abbott, W. D., Dub, F. O., and Nosik, W. A. (1943*a*), "Subdural Hematoma and Effusion as a Result of Blast Injuries. First Preliminary Report", *J.A.M.A.*, 121: 664.

Abbott, W. D., Dub, F. O., and Nosik, W. A. (1943*b*), "Subdural Hematoma and Effusion as a Result of Blast Injuries. Second Preliminary Report. Diagnosis by Psychiatric Examination", *J.A.M.A.*, 121: 739.

Abbott, W. D., Dub, F. O., and Nosik, W. A. (1943*c*), "Subdural Hematoma and Effusion as a Result of Blast Injuries", *Bull. Am. Coll. Surgeons*, 28: 123.

Abbott, W. D., Dub, F. O., and Nosik, W. A. (1943*d*), "Psychiatric Diagnosis of Subdural Hematoma and Effusion from Blast", *Am. J. Psychiat.*, 100: 98.

Brain, W. R., and Cairns, H. (1928), "The Treatment of the Sequels of Head Injury", *Lancet*, 1: 668.

Brain, W. R. (1942), "Rehabilitation after Injuries to Central Nervous System. War Injuries in Relation to Ophthalmology and Neuro-Surgery", *Proc. Roy. Soc. Med.*, 35: 302, 755.

Cairns, H. (1942), "Rehabilitation after Injuries to Central Nervous System", *Proc. Roy. Soc. Med.*, 35: 299.

Da Costa, D. G., and Adson, A. W. (1941), "Subdural Hydroma", *Arch. Surg.*, 43: 559.

Dandy, W. (1932), "Chronic Subdural Hydroma and Serous Meningitis; Pachymeningitis Serosa; Localised External Hydrocephalus", in Lewis's "Practice of Surgery", Prior, Hagerstown, 12: 306.

Ingraham, F. D., and Matson, D. D. (1944), "Subdural Hematoma in Infancy", *J. Pediat.*, 24: 1.

Jefferson, G. (1942), "Rehabilitation after Injuries to Central Nervous System", *Proc. Roy. Soc. Med.*, 35: 295.

Lanigan, J. P. (1944), "Traumatic Subdural Effusion in Children", *Lancet*, 2: 686.

Leary, T., and Edwards, E. A. (1933), "The Subdural Space and its Linings", *Arch. Neurol. & Psychiat.*, 29: 691.

McConnell, A. A. (1941), "Traumatic Subdural Effusions", *J. Neurol., Neurosurg. & Psychiat.*, 4: 237.

McConnell, A. A. (1942), "Intra-Cranial Conditions after Closed Head Injuries", *Brain*, 65: 266.

McConnell, A. A. (1944), "Prolonged Post-Traumatic Amnesia. Findings at Operation", *Lancet*, 1: 278.

McConnell, A. A. (1951), "Pathognomonic Sign of the Leakage of Cerebro-Spinal Fluid into the Subdural Space", *Irish J. M. Sc.*, November: 550.

McConnell, A. A. (1953), "On Certain Sequelæ of Closed Head Injuries. The Pathological Basis of the 'Post-Traumatic Syndrome'", *Brain*, 76: 473.

McConnell, A. A. (1953), "Subdural Air after Pneumoencephalography", *Brain*, 76: 485.

McKay, R. J., junior, Morissette, R. A., Ingraham, F. D., and Matson, D. D. (1950), "Collections of Sub-Dural Fluid Complicating Meningitis due to Hæmophilus Influenzæ (Type B). A Preliminary Report", *New England J. Med.*, 242: 20.

Naffziger, H. C. (1924), "Subdural Fluid Accumulations following Head Injury", *J.A.M.A.*, 82: 1751.

Payr, E. (1916), *"Meningitis serosa bei med nach Schadelverletzungen (traumatica)"*, *Med. Klinik.*, 12: 841.

Penfield, W. G. (1924), "The Cranial Subdural Space (a Method of Study)", *Anat. Rec.*, 28: 173.

Scott, M. (1942), "Prolonged Stupor Produced by Subdural Hygroma", *Am. J. Surg.*, 55: 534.

Smith, M. H. D., Dormont, R. E., and Prather, G. W. (1951), "Subdural Effusions Complicating Bacterial Meningitis", *Pediatrics*, 7: 34.

Steinberg, S. H., and Murphy, J. D. (1951), "Subdural Hygroma Complicating Meningococcal Meningitis", *J. Neurosurg.*, 8: 671.

Symonds, C. P. (1942), "Differential Diagnosis and Treatment of Post-Contusional States", *Proc. Roy. Soc. Med.*, 35: 601.

Trotter, W. (1924), "Certain Minor Injuries of the Brain", *Lancet*, 1: 935.

Trotter, W. (1930), "An Address on Evolution of the Surgery of Head Injuries", *Lancet*, 1: 169.

Walsh, M. N., and Sheldon, C. H. (1937), "Acute Subdural Hydroma: Report of a Case", *Proc. Staff Meet. Mayo Clin.*, 12: 134.

Wycis, H. T. (1945), "Subdural Hygroma", *J. Neurosurg.*, 2: 340.

ATOPIC DERMATITIS (GENERALIZED NEURODERMATITIS): THE ATOPIC DERMATITIS-ASTHMA-HAY FEVER SYNDROME.

By Emil Milder,
Sydney.

In 1892 Besnier described a specific and intensely itchy dermatosis, which was often associated with asthma and hay fever and which occurred—in contrast to the prurigo of Hebra—on the face and the flexor surfaces of the extremities. He stressed the heredity, the nightly paroxysms, the seasonal variations, the chronicity of the disease and the stubbornness to treatment, and separated it strictly from true eczema. He called the disorder *"prurigo diasthésique"*, and expressed the opinion that it might be a variation of *prurigo Hebræ*. Besnier was the first who considered this syndrome from the constitutional-pathological point of view, and stated that the cutaneous changes were due to predisposition, presupposing an abnormal reactivity of the skin.

There is extensive disagreement over the nomenclature, and unfortunately many physicians understand different skin diseases by the terms "generalized or disseminated neurodermatitis", "prurigo Besnier", "atopic dermatitis", etc. This is noticeable sometimes in statistical tabulations, in which figures are given side by side for (*a*) generalized or disseminated neurodermatitis, (*b*) flexural eczema, (*c*) atopic dermatitis, (*d*) prurigo Besnier, etc., even though they are synonyms for the disorder described by Besnier more than six decades ago.

The term "prurigo" is as misleading as is "eczema", because the disorder in fact is neither a prurigo nor an eczema. I shall avoid discussion concerning nomenclature, and in this paper the term "atopic dermatitis" will be used. It must be admitted that this name has been accepted by

a large majority of dermatologists as the most suitable term.

It must be pointed out that this article cannot claim either to survey the voluminous pertinent literature or to give a detailed description of my own cases. In two previous papers (Milder, 1955, 1957) I attempted to stress the influence of neuropsychogenic factors and to discuss the role of allergic phenomena in the ætiology and pathogenesis of the symptom complex. In the present paper I shall merely outline some basic facts and discuss the significance of various factors in connexion with the syndrome, on the basis of my observations and investigations.

The large majority of my cases (84) was chosen from approximately 8000 patients with eczemas and dermatitides (contact eczema, seborrhœic dermatitis, allergic eczematous dermatitis, non-atopic infantile eczema, etc.) in Czechoslovakia (at the Dermatological Department of the State General Hospital in Kosice, in health centres, in semi-military organizations and in private practice). Further cases (11) were found among my patients in New South Wales.

The age incidence was as follows: two months to five years, 22 cases; six to 15 years, 41 cases; 16 to 35 years, 27 cases; 35 to 39 years, five cases; total, 95 cases. Approximately 61% of the patients were males; 36% came from farms and 64% from industrial homes.

Prenatal Influences.

Embryology appears to be the key to the syndrome. The epidermis and the central nervous system develop from the ectoderm, and there exists a very close relationship between the skin, the central nervous system and the endocrine system. These three components of the autonomic system are functionally inseparable, operate correlatively in advanced fœtal life and influence each other mutually. Normally there is complete equilibrium in fœtal life. If this equilibrium is upset in any way, the consequences may be seen either shortly after birth or later in life. It is common knowledge that the disorder does not result from grossly faulty development; but smaller defects or weakness of the ectoderm (Ingram, 1955) may lead to disturbed equilibrium, and may render the subjects over-reactive and susceptible to many stimuli of heterogenous nature. The child with a born imbalance of his autonomous system is thrown into a world full of difficulties, and all his latent predispositions may be mobilized by a number of factors. The disease ranges considerably in distribution, variation and degree of severity, according to which component has been most affected, and there may be a mingling of the types. (Figure I shows a cross-section of the syndrome, the correlation and mutual influence of constitutional and environmental factors.)

An idioplasmic defect or weakness of the ectoderm is generally accepted as the cause of ichthyosis and to a lesser degree of keratotic changes (keratotic pilaris), and it is assumed that they are often predisposing causes of atopic dermatitis. Eye diseases such as keratitis, juvenile cataract and keratoconus, which are occasionally found as complications of atopic dermatitis, are considered additional proof of ectodermal disturbance. In the present series of 95 patients there have been three with ichthyosis and two with keratotic changes, another three with keratitis and two with juvenile cataract.

Endocrine Considerations.

The incidence of atopic dermatitis is correlated with age, and a very considerable percentage of the sufferers experience prolonged remissions or show spontaneous cure after puberty. The exacerbations and remissions of the disease are influenced by certain life phases, and puberty, menstruation, pregnancy and the menopause often determine the severity of the symptoms. Hypogenitalism (small testicles, deficiency of pubic hair, hypoplastic breasts) is a recognized feature. I have found nine males (Figure II) and three females with hypogenitalism in the present series. It may be regarded as a neuro-endocrine (diencephalo-pituitary) dysfunction, occurring in predisposed individuals as a result of endocrine dysequilibrium following genital hypofunction. A precarious equilibrium is established in some, particularly later in life, and prolonged remissions may occur.

Sebaceous and sweat glands are responsive to endocrine stimuli, and the function of these structures is intimately connected with the symptoms of the disease.

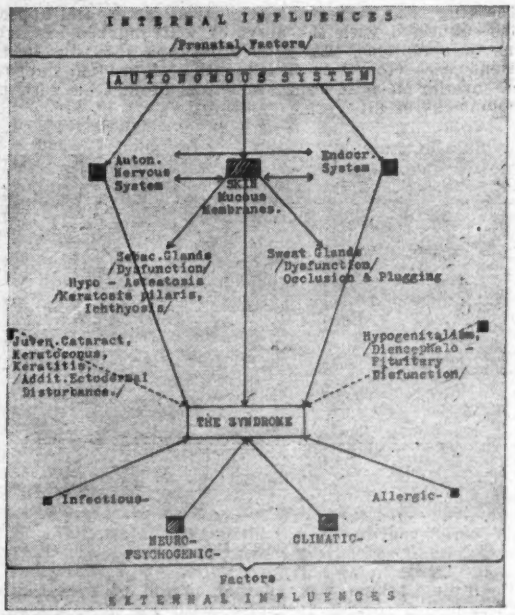


FIGURE I.

According to Selye (personal communication, 1952) there is a "maladaptive factor" at work, and the general pathogenetic interpretation given for all diseases of adaptation could be applied here also.

The concept that atopic dermatitis may be influenced or caused by a derangement of the pituitary-adrenal response to stress is supported by the experiments of Sternberg and Zimmermann (1952), performed on 27 patients suffering from atopic dermatitis. Exposure to extreme heat did not elicit a normal alarm reaction, while epinephrine injections were followed by a normal eosinopenic response. This was interpreted as an indication of normal pituitary function, but as a failure to react to some stimuli.

I should like to quote a statement of Barber (1948) relating to atopic dermatitis:

> These subjects have been investigated *ad nauseam* by different experts, each with his own viewpoint and method of treatment, but they have been neglected by the endocrinologist. Yet a rational explanation of their peculiarities is forthcoming if these are considered in terms of the endocrine-autonomic system, for it is clear, as Bolten (1925) states, that these patients suffer from a "congenital insufficiency" of the sympathetic system and of the endocrine glands with accelerator action! It seems to be likely that the primary defect is an innate hypofunction of the anterior pituitary, which in turn would tend to underaction of the thyroid and adrenal glands and hence of the sympathetic system. The resulting vagotonia accounts for many of their symptoms, and their intolerance of stress (both physical and psychological), their hypotension, their tendency to fluid retention, the biochemical changes that occur during the exacerbation of their symptoms (Barber and Oriel, 1928), their hypersensitiveness to physical and chemical allergens, and their essentially introvert temperament, all are explicable on their basis of this supposition. Furthermore, many exhibit signs of hypogonadism and puberty in both sexes is often delayed.

Though the results of corticotropic and cortisone therapy have been disappointing, I am tempted to think that

hormone therapy may be the treatment of the future. New drugs are under evaluation, and they may radically change all our therapeutic concepts. I shall discuss this later in another paper.

Teething, School Exacerbations, Focal Infections, Climate.

Teething is an important ætiological factor, and appears to be connected with the onset or with marked exacerbations of symptoms. Early teething was the rule in the present cases (fourth to fifth month). A gradual improvement or the onset of remissions was often seen with the development of all teeth.

FIGURE II.

Beginning school was another period of exacerbation, coinciding in several instances with the first attack of asthma. Emotional upsets, such as punishment by the teacher or discrimination by other children, were often the trigger factors. The healthy children often avoided those with skin diseases and excluded them from community games, and the subsequent feeling of inferiority appeared to have an untoward effect upon the course of the disease. A considerable percentage of these children came from broken homes, or had alcoholic fathers. There was a higher incidence among the children of industrial workers, while children of farmers were less often affected (proportion 2:1).

Another cause of exacerbations was upper respiratory infections and chronic bronchitis precipitated attacks of asthma as well as relapses of the cutaneous manifestations. A high incidence of leucocytosis suggested focal infection, and removal of such foci sometimes resulted in remissions and relief of symptoms. The erythrocyte sedimentation rate was in the present cases generally normal, and often vascular hypotension and eosinophilia were present.

All patients were much affected or influenced by climatic conditions. Fog, humidity, sudden fall of barometric pressure and heat caused exacerbations. Most of the patients were under normal weight.

I should like to assume that asthma occurred more often in Australia than in Czechoslovakia as a special consequence of the high humidity. It is the mucous membrane that reacts to stress faster and more frequently. However, the reaction is non-specific.

Sebaceous and Sweat Apparatus.

Dysfunction of the sebaceous gland apparatus is a permanent constitutional feature of these patients.

The principal channels of absorption are the sebaceous glands, and they become smaller and less active over the years. The alterations in the epithelium will therefore play a role of lesser importance than the fact that either the number of sebaceous glands is diminished or these glands are atrophied. The skin remains rough, dry and lustreless. This explains the asteatotic character of the skin, as well as the fact that certain applications—for example, cortisone ointments—work quickly and well in some cases but have a delayed effect in others. Treatment with androgen is often successful, the sebaceous glands becoming more active and larger. I have seen very good results in some cases when androgen was given.

Sweat glands respond to cholinergic stimuli, and a predominantly sympathetic-adrenergic tonus of the skin may minimize vagus stimuli, and thus lead to dysfunction of sweat glands. An abnormal and often absent sweat response is frequently found in these patients, as well as reduced secretion of the sebaceous glands. Some of the present patients became worse during warm summer months owing to œdema of the skin, and I believe that this in consequence led to occlusion of the sweat glands. Sulzberger (1952) describes "plugging and occlusion of the sweat glands over large areas", and refers to a "flaw" in the sweat apparatus and assumes that the temperature-regulating end-organ of the skin may be impaired.

Allergy.

Among sufferers with atopic dermatitis there is a considerable percentage with a family history of so-called "allergic" diseases, like asthma, urticaria, hay fever, infantile eczema, etc. Mostly young people are afflicted. Among the present 95 patients there have been 43 with a relevant family anamnesis.

There is no doubt that allergic sensitization may play some—mostly aggravating—role in infants; but to quote *The Practitioner* (introductory article in the issue of June, 1957), "the number of unfortunate children, for instance, who have been deprived of many of the pleasures of the tea-table because their parents, or doctor, considered them to be 'allergic' to some particularly delectable delicacy must be nearly uncountable".

The followers of the allergic concept stress chiefly the relevance of ingestant, inhalant and autotoxic stimuli, and attribute less significance to other factors. Cutaneous tests, the elimination of contact factors and diets eliminating food allergens, are thought to be of the greatest importance. The presence of antibodies is over-emphasized, and the allergic wheal was considered too loosely as a specific and relevant antigen-antibody reaction.

The antigen-antibody reaction is overwhelmed by factors apart from this reaction (Carryer, 1949) and there is no conclusive proof that the inflammatory lesions are based exclusively on antigen-antibody reactions. It is not possible to discover circulating antibodies in many subjects with clinical allergies, or to give them symptomatic relief by desensitization (H. L. Williams, 1949).

If we cause an experimental skin reaction in a healthy person it is always urticarial, but the inflammatory response in atopic dermatitis goes far beyond a simple urticarial reaction. It is chiefly an irritation of the epithelium leading to acanthotic thickening of the skin, to lichenization (Lutz, 1949). The eczema is superadded. There is a tendency to hyperpigmentation and follicular keratosis.

These patients belong mostly to the group of "physical allergies" (Duke, 1949), and show a sensitivity to physical, thermal, psychogenic and chemical agents—for example, changes in barometric pressure, temperature and humidity —and to emotional disturbances.

I obtained positive reactions in 33 of 53 affected children and in nine of 30 normal control persons. The positive skin reactions found in the present series appeared to be unrelated to the clinical course of the disease, and other allergens seemed to be relevant in only sporadic cases. The elimination of milk or egg from the diet of milk-sensitive or egg-sensitive children or the elimination of other alleged allergens (straw, hay dust, etc.) did not lead to relief of their skin disorder.

The allergic factors may be considered to cause exacerbations in some cases, but the hypersensitivity is of little ætiological importance (Cooke, 1944; Carryer, 1946; Meara, 1955; Milder, 1955, 1957; and others). I had to abandon entirely, as unreliable, intradermal or scratch tests in cases of atopic dermatitis.

It was observed that so-called "eczematous" individuals might have an increased susceptibility to toxic reactions caused by drugs. Neosalvarsan and sulphapyridine may

lead to a toxicoderma of sites of predilection of atopic dermatitis (Milder, 1934, 1945).

I have never been successful with desensitization.

Hyperventilation.

Carryer (1946, 1952) discovered that the loss of carbon dioxide in the alveolar air of the lungs leads to a deficit of alkali and he believes that this transposition may be responsible for clinical symptoms with "relatively unstable patients who are tense, anxious, depressed or psychoneurotic". These people tend to yawn often. His statement corresponds with my observation and with those of schoolteachers and nurses in Czechoslovakia, who reported that children suffering from atopic dermatitis used to yawn a great deal at school and kindergarten, without falling asleep, whenever their skin condition deteriorated.

Pruritus, the Neurovascular Response and the Autonomic Nervous System.

The first and outstanding symptom of atopic dermatitis is pruritus, and most authors agree that this symptom occurs prior to visible cutaneous manifestations, the latter chiefly provoked by scratching. Besnier (1892) wrote: "*Le symptôme premier et le premier symptôme est le prurit intense!*". Kreibich (1927) was of the opinion that the irritation was of a nervous nature, and he called the pruritus occurring in apparently normal skin "fixation phenomenon". Johnson (1951), quoting another author, described the patient as "overdrawn at the bank of nervous energy with the lesions as material security for the overdraft".

The change in permeability as the consequence of increased vascular activity leads to pruritus. Pruritus is considered as a modified pain sensation, and the threshold for pruritus-producing stimuli appears to be low in atopic dermatitis (Cormia, 1947). The itching may be so severe that suicide is not uncommon (Norrlind, 1946).

There are authors who hold the view that asthma is secondary in this syndrome. I am tempted to think that asthma may be as primary as the pruritus of the skin or the hay fever of the nose—in other words, atopic dermatitis is a skin equivalent of asthma and hay fever. The disease as such is the pruritus, which is the primary condition; the eczema is secondary. It is—as mentioned above—only superimposed. All that applies to the skin is also valid for the mucosa of the bronchi and nose, with certain modifications; the mucous membranes react to stimuli faster and more frequently.

An essential hereditary point appears to me to be a profound difference in vascular response, which seems to be present at birth. This fact, as well as the psychic constitution, is of the greatest importance. Stimuli (mostly emotional) react upon the over-sensitive autonomic nervous system, causing vascular disorders and eventually pruritus. These patients show a profound difference in vascular response, and there are mostly irregularity and unevenness of the structure and function of the cutaneous vessels (Müller, 1939, 1949). There is a paradoxical triple response in some atopic dermatitis patients. Instead of an erythema due to axon reflex, there appears a vasoconstriction. The patients show a "white non-urticarial dermographic line", a phenomenon which was also pointed out by Baer (1952). Healthy people and patients with other skin disorders (eczema) show a red dermographic line.

Müller (1939, 1949) believes that patients with atopic dermatitis "may show an inherited tendency" to vascular reactions following stimuli which would not produce pathological changes in normal individuals. To mention one of the papers on this subject, Eyster, Roth and Kierland (1952) studied 24 patients with atopic dermatitis and a series of controls from the point of view of neuro-vascular response. Various stimuli such as cold, fatigue, worry, anger and loss of sleep caused only vasoconstriction in the normal persons, but produced exacerbations in patients with atopic dermatitis. The same authors pointed out that these patients did not show a normal triple response to stroking, but responded with an anæmic zone of vasoconstriction, a white dermographia. This appears to indicate an inherent difference in response in the autonomous nervous systems of these patients.

Psychological Factors.

Plato said: "The great error of our day in the treatment of the human is that physicians separate the soul from the body." I believe that Erasmus Wilson (1856) was one of the first authors who discussed the ætiological importance of emotional factors in certain skin diseases. Other authors —for example, MacKenna, Ingram, Hellier, Wittkower, and many others—indicated the intimate connexion between emotional factors and skin disorders and the fact that psychic conflicts and tension might be reflected on the skin. Florance (1951) had mostly female patients during the last World War, "suffering from the strain of having a husband or son in the forces who had thus overdrawn their nervous energy accounts".

The psychological factor in the ætiology of atopic dermatitis has been underrated in the past, and only during recent years has the association of the "atopic dermatitis-asthma-hay fever syndrome" with neurogenic and psychogenic factors been increasingly recognized. Not only psychiatrists, but also many dermatologists, stress the importance of neuro-psychogenic influences in the ætiology (Ingram, Hellier, MacKenna, and many others). On the other hand, there are authors who are somewhat sceptical about the emotional factors (Sulzberger, O'Leary, Loewenthal and others). Belisario (1952) classifies the dry type of disseminated neurodermatitis (*prurigo diathésique*) as a psychosomatic dermatitis; but like Kelly (1951) he is of the opinion that all other possibilities must be exhausted before a psychosomatic diagnosis is made (1951). Myers (1950) "appeals for a compromise between the rationalism of Virchow and the idealism of Coué", and holds that in certain cases psychiatric consultation is indicated.

D. H. Williams (1951) found the so-called "maternal rejection factor" as a frequently present character trend in adult atopic dermatitis. In a previous paper (Milder, 1955), two such case reports were chosen to demonstrate this "repressed hostility" against the mother.

Atopic dermatitis is often found in families in which nervous parents or relatives constantly worry about the patient, talk much about their respective diseases, etc., causing increased irritability of the whole autonomic nervous system.

Discussion.

There is sufficient evidence to assume that atopic dermatitis is an hereditary disease and that the predisposition is present at birth. The syndrome may be caused by developmental defects or weakness of the ectoderm. Such ectodermal defects or weakness may influence the three components of the autonomic system to different extents and predispose the subject to disturbances of the skin and mucous membranes. The autonomic system appears to be impaired, and as the components of this system are interrelated, different forms of the disease occur, according to which component has suffered most. There is "gradual rather than abrupt transition from one type to the other and there may be a mingling of the types" (H. L. Williams, 1949).

The congenital hypersensitivity of the total organism may not become apparent immediately after the first contact or after the first "irritation"; this altered reactivity may be observed after repeated irritation only. In addition to this hypersensitivity, the respiratory organs, the skin (part of the vascular apparatus of the skin), the mucous membranes of the bronchial tubes and the nose, are ready to react with pathological changes to stimuli which are well tolerated by normal subjects.

In general it may be said that the patients show lability of the autonomic nervous system and have a certain constitutional type. I think that the psychological factors are more important than the allergic findings in these patients. It appears to be a question of time and conditions when the latent predisposition becomes manifest and when the first signs occur. I believe that even in childhood psychological stimuli occur more often and are of greater importance than it has been assumed before, and that infections, and ingestant, inhalant and autotoxic stimuli are of much less importance than emotional factors. The fact that a change of environment, even without admission to hospital and medicines, leads to improvement is in favour of that conception.

hormone therapy may be the treatment of the future. New drugs are under evaluation, and they may radically change all our therapeutic concepts. I shall discuss this later in another paper.

Teething, School Exacerbations, Focal Infections, Climate.

Teething is an important ætiological factor, and appears to be connected with the onset or with marked exacerbations of symptoms. Early teething was the rule in the present cases (fourth to fifth month). A gradual improvement or the onset of remissions was often seen with the development of all teeth.

FIGURE II.

Beginning school was another period of exacerbation, coinciding in several instances with the first attack of asthma. Emotional upsets, such as punishment by the teacher or discrimination by other children, were often the trigger factors. The healthy children often avoided those with skin diseases and excluded them from community games, and the subsequent feeling of inferiority appeared to have an untoward effect upon the course of the disease. A considerable percentage of these children came from broken homes, or had alcoholic fathers. There was a higher incidence among the children of industrial workers, while children of farmers were less often affected (proportion 2:1).

Another cause of exacerbations was upper respiratory infections and chronic bronchitis precipitated attacks of asthma as well as relapses of the cutaneous manifestations. A high incidence of leucocytosis suggested focal infection, and removal of such foci sometimes resulted in remissions and relief of symptoms. The erythrocyte sedimentation rate was in the present cases generally normal, and often vascular hypotension and eosinophilia were present.

All patients were much affected or influenced by climatic conditions. Fog, humidity, sudden fall of barometric pressure and heat caused exacerbations. Most of the patients were under normal weight.

I should like to assume that asthma occurred more often in Australia than in Czechoslovakia as a special consequence of the high humidity. It is the mucous membrane that reacts to stress faster and more frequently. However, the reaction is non-specific.

Sebaceous and Sweat Apparatus.

Dysfunction of the sebaceous gland apparatus is a permanent constitutional feature of these patients.

The principal channels of absorption are the sebaceous glands, and they become smaller and less active over the years. The alterations in the epithelium will therefore play a role of lesser importance than the fact that either the number of sebaceous glands is diminished or these glands are atrophied. The skin remains rough, dry and lustreless. This explains the asteatotic character of the skin, as well as the fact that certain applications—for example, cortisone ointments—work quickly and well in some cases but have a delayed effect in others. Treatment with androgen is often successful, the sebaceous glands becoming more active and larger. I have seen very good results in some cases when androgen was given.

Sweat glands respond to cholinergic stimuli, and a predominantly sympathetic-adrenergic tonus of the skin may minimize vagus stimuli, and thus lead to dysfunction of sweat glands. An abnormal and often absent sweat response is frequently found in these patients, as well as reduced secretion of the sebaceous glands. Some of the present patients became worse during warm summer months owing to œdema of the skin, and I believe that this in consequence led to occlusion of the sweat glands. Sulzberger (1952) describes "plugging and occlusion of the sweat glands over large areas", and refers to a "flaw" in the sweat apparatus and assumes that the temperature-regulating end-organ of the skin may be impaired.

Allergy.

Among sufferers with atopic dermatitis there is a considerable percentage with a family history of so-called "allergic" diseases, like asthma, urticaria, hay fever, infantile eczema, etc. Mostly young people are afflicted. Among the present 95 patients there have been 43 with a relevant family anamnesis.

There is no doubt that allergic sensitization may play some—mostly aggravating—role in infants; but to quote *The Practitioner* (introductory article in the issue of June, 1957), "the number of unfortunate children, for instance, who have been deprived of many of the pleasures of the tea-table because their parents, or doctor, considered them to be 'allergic' to some particularly delectable delicacy must be nearly uncountable".

The followers of the allergic concept stress chiefly the relevance of ingestant, inhalant and autotoxic stimuli, and attribute less significance to other factors. Cutaneous tests, the elimination of contact factors and diets eliminating food allergens, are thought to be of the greatest importance. The presence of antibodies is over-emphasized, and the allergic wheal was considered too loosely as a specific and relevant antigen-antibody reaction.

The antigen-antibody reaction is overwhelmed by factors apart from this reaction (Carryer, 1949) and there is no conclusive proof that the inflammatory lesions are based exclusively on antigen-antibody reactions. It is not possible to discover circulating antibodies in many subjects with clinical allergies, or to give them symptomatic relief by desensitization (H. L. Williams, 1949).

If we cause an experimental skin reaction in a healthy person it is always urticarial, but the inflammatory response in atopic dermatitis goes far beyond a simple urticarial reaction. It is chiefly an irritation of the epithelium leading to acanthotic thickening of the skin, to lichenization (Lutz, 1949). The eczema is superadded. There is a tendency to hyperpigmentation and follicular keratosis.

These patients belong mostly to the group of "physical allergies" (Duke, 1949), and show a sensitivity to physical, thermal, psychogenic and chemical agents—for example, changes in barometric pressure, temperature and humidity —and to emotional disturbances.

I obtained positive reactions in 33 of 53 affected children and in nine of 30 normal control persons. The positive skin reactions found in the present series appeared to be unrelated to the clinical course of the disease, and other allergens seemed to be relevant in only sporadic cases. The elimination of milk or egg from the diet of milk-sensitive or egg-sensitive children or the elimination of other alleged allergens (straw, hay dust, etc.) did not lead to relief of their skin disorder.

The allergic factors may be considered to cause exacerbations in some cases, but the hypersensitivity is of little ætiological importance (Cooke, 1944; Carryer, 1946; Meara, 1955; Milder, 1955, 1957; and others). I had to abandon entirely, as unreliable, intradermal or scratch tests in cases of atopic dermatitis.

It was observed that so-called "eczematous" individuals might have an increased susceptibility to toxic reactions caused by drugs. Neosalvarsan and sulphapyridine may

lead to a toxicoderma of sites of predilection of atopic dermatitis (Milder, 1934, 1945).

I have never been successful with desensitization.

Hyperventilation.

Carryer (1946, 1952) discovered that the loss of carbon dioxide in the alveolar air of the lungs leads to a deficit of alkali and he believes that this transposition may be responsible for clinical symptoms with "relatively unstable patients who are tense, anxious, depressed or psychoneurotic". These people tend to yawn often. His statement corresponds with my observation and with those of schoolteachers and nurses in Czechoslovakia, who reported that children suffering from atopic dermatitis used to yawn a great deal at school and kindergarten, without falling asleep, whenever their skin condition deteriorated.

Pruritus, the Neurovascular Response and the Autonomic Nervous System.

The first and outstanding symptom of atopic dermatitis is pruritus, and most authors agree that this symptom occurs prior to visible cutaneous manifestations, the latter chiefly provoked by scratching. Besnier (1892) wrote: "*Le symptôme premier et le premier symptôme est le prurit intense!*". Kreibich (1927) was of the opinion that the irritation was of a nervous nature, and he called the pruritus occurring in apparently normal skin "fixation phenomenon". Johnson (1951), quoting another author, described the patient as "overdrawn at the bank of nervous energy with the lesions as material security for the overdraft".

The change in permeability as the consequence of increased vascular activity leads to pruritus. Pruritus is considered as a modified pain sensation, and the threshold for pruritus-producing stimuli appears to be low in atopic dermatitis (Cormia, 1947). The itching may be so severe that suicide is not uncommon (Norrlind, 1946).

There are authors who hold the view that asthma is secondary in this syndrome. I am tempted to think that asthma may be as primary as the pruritus of the skin or the hay fever of the nose—in other words, atopic dermatitis is a skin equivalent of asthma and hay fever. The disease as such is the pruritus, which is the primary condition; the eczema is secondary. It is—as mentioned above—only superimposed. All that applies to the skin is also valid for the mucosa of the bronchi and nose, with certain modifications; the mucous membranes react to stimuli faster and more frequently.

An essential hereditary point appears to me to be a profound difference in vascular response, which seems to be present at birth. This fact, as well as the psychic constitution, is of the greatest importance. Stimuli (mostly emotional) react upon the over-sensitive autonomic nervous system, causing vascular disorders and eventually pruritus. These patients show a profound difference in vascular response, and there are mostly irregularity and unevenness of the structure and function of the cutaneous vessels (Müller, 1939, 1949). There is a paradoxical triple response in some atopic dermatitis patients. Instead of an erythema due to axon reflex, there appears a vasoconstriction. The patients show a "white non-urticarial dermographic line", a phenomenon which was also pointed out by Baer (1952). Healthy people and patients with other skin disorders (eczema) show a red dermographic line.

Müller (1939, 1949) believes that patients with atopic dermatitis "may show an inherited tendency" to vascular reactions following stimuli which would not produce pathological changes in normal individuals. To mention one of the papers on this subject, Eyster, Roth and Kierland (1952) studied 24 patients with atopic dermatitis and a series of controls from the point of view of neuro-vascular response. Various stimuli such as cold, fatigue, worry, anger and loss of sleep caused only vasoconstriction in the normal persons, but produced exacerbations in patients with atopic dermatitis. The same authors pointed out that these patients did not show a normal triple response to stroking, but responded with an anæmic zone of vasoconstriction, a white dermographia. This appears to indicate an inherent difference in response in the autonomous nervous systems of these patients.

Psychological Factors.

Plato said: "The great error of our day in the treatment of the human is that physicians separate the soul from the body." I believe that Erasmus Wilson (1856) was one of the first authors who discussed the ætiological importance of emotional factors in certain skin diseases. Other authors —for example, MacKenna, Ingram, Hellier, Wittkower, and many others—indicated the intimate connexion between emotional factors and skin disorders and the fact that psychic conflicts and tension might be reflected on the skin. Florance (1951) had mostly female patients during the last World War, "suffering from the strain of having a husband or son in the forces who had thus overdrawn their nervous energy accounts".

The psychological factor in the ætiology of atopic dermatitis has been underrated in the past, and only during recent years has the association of the "atopic dermatitis-asthma-hay fever syndrome" with neurogenic and psychogenic factors been increasingly recognized. Not only psychiatrists, but also many dermatologists, stress the importance of neuro-psychogenic influences in the ætiology (Ingram, Hellier, MacKenna, and many others). On the other hand, there are authors who are somewhat sceptical about the emotional factors (Sulzberger, O'Leary, Loewenthal and others). Belisario (1952) classifies the dry type of disseminated neurodermatitis (*prurigo diathésique*) as a psychosomatic dermatitis; but like Kelly (1951) he is of the opinion that all other possibilities must be exhausted before a psychosomatic diagnosis is made (1951). Myers (1950) "appeals for a compromise between the rationalism of Virchow and the idealism of Coué", and holds that in certain cases psychiatric consultation is indicated.

D. H. Williams (1951) found the so-called "maternal rejection factor" as a frequently present character trend in adult atopic dermatitis. In a previous paper (Milder, 1955), two such case reports were chosen to demonstrate this "repressed hostility" against the mother.

Atopic dermatitis is often found in families in which nervous parents or relatives constantly worry about the patient, talk much about their respective diseases, etc., causing increased irritability of the whole autonomic nervous system.

Discussion.

There is sufficient evidence to assume that atopic dermatitis is an hereditary disease and that the predisposition is present at birth. The syndrome may be caused by developmental defects or weakness of the ectoderm. Such ectodermal defects or weakness may influence the three components of the autonomic system to different extents and predispose the subject to disturbances of the skin and mucous membranes. The autonomic system appears to be impaired, and as the components of this system are interrelated, different forms of the disease occur, according to which component has suffered most. There is "gradual rather than abrupt transition from one type to the other and there may be a mingling of the types" (H. L. Williams, 1949).

The congenital hypersensitivity of the total organism may not become apparent immediately after the first contact or after the first "irritation"; this altered reactivity may be observed after repeated irritation only. In addition to this hypersensitivity, the respiratory organs, the skin (part of the vascular apparatus of the skin), the mucous membranes of the bronchial tubes and the nose, are ready to react with pathological changes to stimuli which are well tolerated by normal subjects.

In general it may be said that the patients show lability of the autonomic nervous system and have a certain constitutional type. I think that the psychological factors are more important than the allergic findings in these patients. It appears to be a question of time and conditions when the latent predisposition becomes manifest and when the first signs occur. I believe that even in childhood psychological stimuli occur more often and are of greater importance than it has been assumed before, and that infections, and ingestant, inhalant and autotoxic stimuli are of much less importance than emotional factors. The fact that a change of environment, even without admission to hospital and medicines, leads to improvement is in favour of that conception.

Individual differences in the reactions may be found in every age group, and the clinical manifestations of different forms may be watched during infancy, during childhood, during puberty and in adult age groups. It is preferable to use the term "infantile eczema" in the first two years of life, because an early manifestation of atopic dermatitis cannot be diagnosed with certainty.

The differences in the clinical picture in various age groups may be due to hereditary and constitutional factors, to different properties in the skin, the vascular and nervous system and the endocrine apparatus, and to environmental factors. It is difficult to explain why irritations—external or internal—may cause manifestations on different areas of the body at a certain age, or why irritations which operate in infants are ineffective in adult life and vice versa. I cannot explain why atopic dermatitis becomes worse and the asthma is relieved, or why there are cases in which both appear at the same time.

Like many others, I have noted a connexion between the beginning or exacerbation of the disease and the dentition. I have observed a gradual improvement after all the deciduous teeth have developed, a proof that the "irritation" has ceased.

The following causes of the manifestation or exacerbation seem to be of considerable importance: anxiety and overstimulation; exciting events; events connected with school (beginning at school, excitement at games, punishment, etc.); "maternal rejecting factor"; emotional upsets; etc.

In general I agree with those authors who find skin tests with foodstuffs an impracticable method, and employ a course of simple psychotherapy with sympathy, patience and understanding—an attempt to relieve the tension by permitting the patient to talk and to express himself. "Each and every patient must be considered as a separate and individual problem" (Kelly, 1951). The cases become clearer if it is possible to eliminate one or the other pathogenetic factor (Lutz, 1949).

Conclusions and Summary.

Ninety-five cases of atopic dermatitis in different age groups have been observed and investigated, and the significance of various factors has been discussed. The present knowledge of the "atopic dermatitis-asthma-hay fever" problem permits the following conclusions to be drawn:

1. The syndrome is a distinct clinical entity and is hereditary.

2. The patients represent a special constitutional type, and show an abnormal reaction to a great variety of stimuli.

3. The abnormal reactivity appears to be due to prenatal influences (ectodermal defect or weakness).

4. Emotional stimuli occur frequently and are of the greatest importance.

5. There is a profound difference in vascular response.

6. Tests of inhalant and ingestant allergens are of no value in adults and of problematical value in children.

7. There is a strong endocrine influence. The incidence and severity of symptoms decrease with age.

8. There is an abnormal sweat response, with hypofunction of the sebaceous gland apparatus.

References.

BAER, R. L. (1952), discussion following paper by Eyster, W. H., Roth, G. M., and Kierland, R. R., "Studies on the Peripheral Vascular Physiology of Patients with Atopic Dermatitis", *J. Invest. Dermat.*, 18: 37.

BARBER, H. W. (1948), quoted in MacKenna, R. M. B. (1948), *loco citato*.

BELISARIO, J. C. (1951), discussion following paper by Kelly, J. H., "Psychosomatic Factors in Dermatology", *Australian J. Dermat.*, 1: 58.

BELISARIO, J. C. (1952), "The Pathogenesis of Eczema. I", Proceedings of the Tenth International Congress of Dermatology, London, B.M.A.

BESNIER, E. (1892), *"Première note et observations préliminaires pour servir d'introduction à l'étude des prurigos diathésiques (dermatites multiformes prurigineuses chroniques et paroxystiques du type de prurigo de Hebra)". Ann. de dermat. et syph.*, page 634.

CARRYER, H. M. (1946), "The Role of Hyperventilation in Functional Disorders", *Proc. Staff Meet., Mayo Clin.*, 21: 361.

CARRYER, H. M. (1952), "Hyperventilation Syndrome", *Proc. Staff Meet., Mayo Clin.*, 27: 58.

COOKE, J. (1944), "A Consideration of Allergic Problems", *J. Allergy*, 15: 203.

CORMIA, F. E. (1947), "Psychosomatic Factors in Dermatoses. A Critical Analysis of Diagnostic Methods of Approach", *Arch. Dermat.*, 55: 601.

DUKE, W. W. (1949), quoted by Williams, H. L., *loco citato*.

EYSTER, W. H., ROTH, G. M., and KIERLAND, R. R. (1952), "Studies of the Peripheral Vascular Physiology of Patients with Atopic Dermatitis", *J. Invest. Dermat.*, 18: 37.

FLORANCE, F. C. (1951), discussion following paper by Kelly, J. H., "Psychosomatic Factors in Dermatology", *Australian J. Dermat.*, 1: 58.

HELLIER, F. F. (1948), quoted by Wittkower, E., in MacKenna, R. M. B. (1948), *loco citato*.

INGRAM, J. T. (1955), "Besnier's Prurigo: An Ectodermal Defect", *Brit. J. Dermat.*, 67: 43.

JOHNSON, A. M. (1951), quoted by Florance, F. C., *Australian J. Dermat.*, 1: 58.

KELLY, J. H. (1951), "Psychosomatic Factors in Dermatology", *Australian J. Dermat.*, 1: 39.

KREIBICH, C. (1927), *"Ekzeme und Dermatitiden"*, in Jadassohn, J. (Editor), *Handbuch der Haut- und Geschlechtskrankheiten*", Springer, Berlin, Volume VI.

LUTZ, W. (1949), "Neurodermatitis (Prurigo, Ekzem)", *Dermatologica*, 98, 1.

MACKENNA, R. M. B. (1944), "Psychosomatic Factors in Cutaneous Diseases", *Lancet*, 2: 679.

MACKENNA, R. M. B. (1948) (editor), "Modern Trends in Dermatology", Butterworth, London.

MEARA, R. H. (1955), "Skin Reactions in Atopic Eczema", *Brit. J. Dermat.*, 67: 60.

MILDER, E. (1934), *"Neosalvarsandermatitis mit beiderseitiger Parotitis", Dermat. Wchnschr.*, 38: 1245.

MILDER, E. (1945), paper read at clinical meeting, State General Hospital, Kosice, Czechoslovakia.

MILDER, E. (1955), "The Influence of Neuro-Psychogenic Factors in the Etiology of Generalized Neurodermatitis", *Dermatologica*, 111: 140.

MILDER, E. (1957), "The Role of Allergic Phenomena in the Etiology and Pathogenesis of Atopic Dermatitis", *Arch. Dermat.*, 76: 27.

MÜLLER, O. (1939), *"Die feinsten Blutgefässe des Menschen in gesunden und kranken Tagen"*, Enke, Stuttgart, Volume 2.

MÜLLER, O. (1949), quoted by Williams, H. L., *loco citato*.

MYERS, W. K. (1951), "The Psychosomatic Aspects of Skin Diseases", M. J. AUSTRALIA, 1: 476.

NORRLIND, R. (1946), "Prurigo Besnier, Atopic Dermatitis", *Acta derm.-venereol.*, 26, Suppl. XIII.

SELYE, H. (1952), personal communication.

STERNBERG, T. H., and ZIMMERMANN, M. C. (1952), "Stress Studies in the Eczema-Asthma-Hay Fever Diathesis", *Arch. Dermat.*, 65: 392.

SULZBERGER, M. B. (1940), "Dermatologic Allergy", Springfield, Thomas.

SULZBERGER, M. B. (1952), *Arch. Dermat.*, 65: 392.

WILLIAMS, D. H. (1951), "Management of Atopic Dermatitis in Children, Control of the Maternal Rejection Factor", *Arch. Dermat.*, 63: 545.

WILLIAMS, H. L. (1949), "A Phylogenetic Concept of Allergy", *Proc. Staff Meet. Mayo Clin.*, 24: 516.

WILSON, E. (1856), "Diseases of the Skin", 4th Edition, London.

WITTKOWER, E. (1948), "Psychological Aspects of Dermatology", in MacKenna, R. M. B. (1948), loco citato.

SERUM GLUTAMIC OXALACETIC-TRANSAMINASE ACTIVITY IN THE DIAGNOSIS OF MYOCARDIAL INFARCTION AND HEPATIC NECROSIS.

By M. N. I. WALTERS,

Department of Medicine, University of Western Australia,

AND

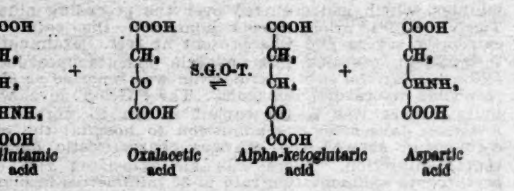
PATRICIA G. LITTLEJOHN,

Biochemistry Department, Royal Perth Hospital, Perth.

SINCE the observation that raised levels of the enzyme glutamic oxalacetic transaminase were found in blood serum after myocardial infarction (La Due, Wroblewski and La Due, 1954) and liver cell damage (Wroblewski and Karmen, 1954), numerous reports on the diagnostic value of serum glutamic oxalacetic transaminase (S.G.O.T.) estimations have appeared in the American literature. An excellent review of the present position in this sphere of clinical enzymology has recently appeared (Mason and Wroblewski, 1957).

Transamination consists of the enzyme-catalysed reversible transfer of the alpha amino-nitrogen of an amino acid to an alpha keto acid, resulting in the

synthesis of a second amino acid and a second alpha keto acid.



The two transaminases of present clinical interest are glutamic-oxalacetic (G.O-T.) and glutamic-pyruvic (G.P-T.).

G.O-T.

(a) Glutamic acid + oxalacetic acid $\rightleftharpoons$ α-ketoglutaric acid + aspartic acid.

G.P-T.

(b) Glutamic acid + pyruvic acid $\rightleftharpoons$ α-ketoglutaric acid + alanine.

The enzymes may be estimated chromatographically (Karmen, Wroblewski and La Due, 1955), spectrophotometrically (Karmen, 1955) or colorimetrically (Cabaud, Leeper and Wroblewski, 1957).

Awapara and Seale in 1952 described transaminase activities in homogenates of skeletal muscle, liver, heart, kidney, spleen, ventral prostate, brain and testis of male rats; however, the activity varied considerably in each of the organs studied. In man, in decreasing order of concentration, the distribution was in the heart, liver, skeletal muscle and kidney. The presence of the glutamic-oxalacetic (G.O-T.) and the glutamic-pyruvic (G.P-T.) transaminase systems in serum of normal individuals was described by Karmen, Wroblewski and La Due (1955).

It has been postulated that as the enzyme is normally intracellular with a low serum titre, necrosis of enzyme-rich cells might result in the transient liberation of excess G.O-T. into the circulation. The consistency of S.G.O-T. elevation following myocardial necrosis has indeed been demonstrated (Nydick *et alii*, 1957). In experimental transmural myocardial infarction, S.G.O-T. elevation occurred approximately six to twelve hours after the occlusion. During the first 48 hours the level rose to a peak, and over the subsequent three to six days gradually returned to the normal range (La Due, Wroblewski and Karmen, 1954).

Elevation of S.G.O-T. levels has been shown to be of considerable diagnostic importance in myocardial infarction in cases in which electrocardiographic tracings are unhelpful, owing to the presence of patterns of left bundle branch block, Wolff-Parkinson-White syndrome or past ischæmic change (Krause and Krause, 1956).

Elevation of S.G.O-T. levels following hepato-cellular injury has been shown to be proportional to the extent of hepatic parenchymal necrosis (Molander, Wroblewski and La Due, 1955). Elevated values were found in viral hepatitis, after carbon tetrachloride intoxication or obstruction to the biliary tract, and in primary and secondary carcinomata of the liver (Chinsky, Schmagranoff and Sherry, 1956). Significant elevation of the S.G.O-T. level has also been described in salicylate intoxication, and it is thought that this is the cause of the occasional abnormal levels found in rheumatic carditis.

Determinations of S.G.O-T. levels in numerous pathological states have been made, and in summary Mason and Wroblewski state that "in the absence of injury to the heart, liver, skeletal muscle and kidney tissue, no significant alterations in S.G.O-T. activity have been observed in neoplastic, allergic, infectious, congenital, degenerative, metabolic, and reactive or collagen disease states".

A more recent application of the study of the serum transaminase levels in the diagnosis of hepatic disease has appeared in the Italian literature (de Ritis, Coltorti and Giusti, 1955). These investigators studied G.O-T. and G.P-T. in sera of patients with viral hepatitis, and found the ratio G.O-T./G.P-T. to be less than unity. The ratio was greater than unity in normal serum and in patients with uncomplicated obstructive biliary disease.

In 1957, however, Wroblewski and Cabaud made estimations of both enzymes in a series of patients suffering from infectious hepatitis, homologous serum hepatitis and biliary obstruction from a variety of etiological agents. Calculation of G.O-T./G.P-T. ratios from a study of these figures showed no obvious differentiation between viral hepatitis and biliary obstruction.

The purpose of this communication is to record data obtained from S.G.O-T. estimations in 60 cases of cardiovascular and hepatic disease at the Royal Perth Hospital.

METHODS.

The following were the methods used for the estimation of S.G.O-T.

Spectrophotometric Method (Karmen, 1955).

Reagents.

The following reagents were used:

(i) Phosphate buffer, 0·1 M, pH 7·4. (ii) Aspartic acid, 0·2 M; a solution of 1·331 grammes of aspartic acid in 35 millilitres of phosphate buffer was adjusted to pH 7·4 (glass electrode) with normal sodium hydroxide solution (approximately 10 millilitres). The final volume was made up to 50 millilitres with buffer. (iii) Alpha ketoglutaric acid, 0·1 M: a solution of 0·735 gramme of alpha ketoglutaric acid in 35 millilitres of phosphate buffer was adjusted to pH 7·4 with sodium hydroxide (approximately 10 millilitres). The final volume was made up to 50 millilitres with buffer. (iv) Reduced diphosphopyridine nucleotide (DPNH), obtained from Sigma Chemical Company: 1·0 milligramme was allowed per millilitre of buffer and prepared freshly each day. (v) Malic dehydrogenase: this was obtained in solution from Sigma Chemical Company, and contained 10,000 units per millilitre. One unit of malic dehydrogenase activity was defined as that amount which decreases optical density at 340 millimicrons by 0·001 per minute in the reaction

$$\text{Oxalacetate} + \text{DPNH} + \text{H}^+ \xrightleftharpoons{\text{malic dehydrogenase}} \text{Malate} + \text{DPN}^+$$

during the initial period while the reaction is still linear with time. A concentration of 2000 units per millilitre was used.

Procedure.

The procedure was as follows.

For the test solution 1·0 millilitre of serum, 1·0 millilitre of phosphate buffer, 0·5 millilitre of 0·2 M aspartic acid, 0·2 millilitre of DPNH and 0·1 millilitre of malic dehydrogenase were pipetted and mixed in a silica cuvette with one centimetre light path. The blank contained all reagents except DPNH, the volume being adjusted with 0·2 millilitre of phosphate buffer. The mixtures were allowed to stand for 10 minutes at room temperature, then 0·2 millilitre of alpha ketoglutaric acid solution was added to both cuvettes and mixed. The optical density at 340 millimicrons was followed in the test solution for five minutes by means of a Hilger Uvispek set with blank solution.

The rate of decrease of optical density was taken as the measure of transaminase activity of the serum. The activity is expressed in units per millilitre of serum per minute, one unit being a decrease in optical density of 0·001 under the conditions described. For very high values a reduced amount of serum was used and the appropriate calculations were made.

Colorimetric Method.

The colorimetric method (Leitman and Frankel, quoted by Sigma, 1956) was used in preference to the spectrophotometric method, as reagents were more easily obtainable and the estimation was more easily performed.

Reagents.

The reagents (as supplied by Sigma Chemical Company) were as follows:

(i) Aspartic acid-alpha ketoglutaric acid substrate, pH 7·5. (ii) Colour reagent: 2,4-dinitrophenylhydrazine and hydrochloric acid. (iii) Standard oxalacetic acid solution. (iv) Sodium hydroxide, 0·4 N.

Procedure.

The procedure was as follows:

(1) 0·2 millilitre of serum was added to 1·0 millilitre of aspartic acid-alpha ketoglutaric acid, already at 37° C.; (ii) exactly 60 minutes after the addition of serum, 1·0 millilitre

of colour reagent was added, and the solution was mixed and left at room temperature; (iii) 20 minutes after step (ii), 10 millilitres of 0·4 N. sodium hydroxide solution were added, and mixing was performed by inversion, clean rubber stoppers being used; (iv) exactly 30 minutes after step (iii), a reading was made on the absorptiometer (Spekker) at 505 millimicrons approximately, the instrument being set with a water blank.

The units of transaminase activity are measured as microgrammes of oxalacetic acid produced per millilitre of serum per minute. The value is read from an oxalacetic acid standard curve.

Normal Figures.

Karmen (1955) found the concentration of S.G.O-T. to lie within the range 9·0 to 32 units per millilitre per minute. Values for normal serum fell within this range with either the spectrophotometric method (10 sera) or the colorimetric method (seven sera).

CLINICAL MATERIAL.

One or more estimations of S.G.O-T. have been performed on 60 patients admitted to the Royal Perth Hospital during late 1956 and the beginning of 1957. The emphasis has been naturally on cases of suspected myocardial infarction and hepatic injury.

RESULTS.

Patients with Suspected Myocardial Disease.

The results in the first group are shown in Table I. Of the 13 cases of proven infarction, the S.G.O-T. level was elevated in nine. Of the four cases in which the reading proved normal, in three estimations were performed at least one week after the acute attack of pain. As has previously been indicated, S.G.O-T. levels in myocardial infarction revert to normal within three to six days of the occlusion. In the remaining case no explanation of the normal result is offered, except that unfortunately only one estimation was performed, and it is presumed that infarction had not occurred at the time of venepuncture, the pain being due to coronary insufficiency only.

TABLE I.

Showing S.G.O-T. Levels in 41 Cases of Suspected Cardiac Disease.

Disease.	Number of Cases.	S.G.O-T. Level Elevated.	Range Units.
Infarction	13	9	21–220
Ischæmia but no infarction	18	—	5–37
Pericarditis	2	—	13–20
Acute rheumatism ..	1	—	22
Myxœdema	1	—	40
Myocarditis	1	1	49
Functional	3	—	5–13
Miscellaneous:			
Hiatus hernia	1	—	20
Carcinomatous pleurisy	1	—	14

It was found that in only two cases were the electrocardiographic changes diagnostic of infarction and the leucocyte count, erythrocyte sedimentation rate and S.G.O-T. levels all elevated. In only two cases were the electrocardiographic and S.G.O-T. readings both diagnostic of infarction. In five cases, therefore, the diagnosis of myocardial infarction was made because of elevation of the S.G.O-T. level coupled with historical and physical evidence (with or without changes in the erythrocyte sedimentation rate and leucocyte count) either at the bedside or at autopsy (see Table II).

In 18 cases presenting with precordial pain of cardiac origin, the electrocardiogram presented evidence of acute coronary insufficiency, but no evidence of infarction. In none of these cases was the S.G.O-T. level raised.

CASE I.—A man, aged 51 years, was admitted to hospital suffering from attacks of substernal pain lasting up to 30 minutes, which had occurred over the preceding nine days. Twelve hours prior to his admission the pain became extremely severe and was present at rest. Examination of the patient revealed mild pyrexia and tachycardia. The following day the electrocardiogram was reported as showing "probable myocardial ischæmia". The S.G.O-T. level was 177 units. There was a subsequent return to normal levels. Eighteen days after his admission to hospital the electrocardiogram showed the changes characteristic of anteroseptal infarction. There was an associated rise of the erythrocyte sedimentation rate to 30 millimetres in one hour, although no leucocytosis was observed. (See Figure I.)

TABLE II.

Showing the Nine Cases of Infarction in which the S.G.O-T. Level was Elevated, Correlated with Routine Investigations—Leucocyte Count, Erythrocyte Sedimentation Rate and Electrocardiogram.

Number of Cases.	History.	Electrocardiographic Findings.	Increase in Leucocyte Count.	Increased Erythrocyte Sedimentation Rate.	Increased S.G.O-T. Level.
2	+	+	+	+	+
2	+	+	−	−	+
5	+	−	±	±	+

CASE II.—A man, aged 35 years, was admitted to hospital because of substernal pain of seven hours' duration, unrelieved by morphine or pethidine. The pain was made worse by respiratory movement. Physical examination of the patient revealed pyrexia, tachycardia and a cold greyish skin. No friction rub was heard. Serial electrocardiograms showed a pattern consistent with a diagnosis of pericarditis. On three occasions the S.G.O-T. level was found to be within the normal range. At no time was any elevation of the erythrocyte sedimentation rate or leucocytosis observed. A clinical diagnosis of acute benign pericarditis was made. After 11 days of supportive treatment the patient was discharged from hospital.

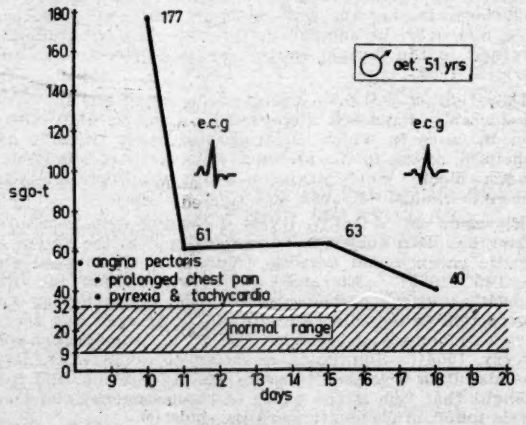


FIGURE I.

Chart of case of myocardial infarction showing serial S.G.O-T levels.

CASE III.—A Southern European man, aged 53 years, had been diagnosed as having a "bad heart" for three years because of the occurrence of precordial pain associated with exertion. Three days prior to his admission to hospital the patient suffered a further attack of pain in the chest and upper part of the abdomen, which radiated to the left shoulder and neck; it was present at rest and lasted some hours. No significant physical abnormalities were found. The electrocardiogram was consistent with the picture of an old myocardial infarction, but the S.G.O-T. level was normal (20 units). After nine days' treatment with coronary dilator drugs, he was discharged from hospital. A subsequent barium meal X-ray examination revealed a reducible hiatus hernia.

TABLE III.
Showing S.G.O-T. Levels in 19 Cases of Suspected Hepatic Disease.

Diagnosis.	Number of Cases.	Results of Liver Function Tests.		S.G.O-T. Level.			Range Units.
		Abnormal.	Normal.	Abnormal.	Borderline.	Normal.	
Inflammatory conditions:							
Viral	3	2	1	3	—	—	62–430
Pyæmic abscess	1	1	—	1	—	—	180
Cholangitis	2	2	—	2	—	—	52–110
Cirrhosis (alcoholic etc.)	6	2	4	3	2	1	18–362
Neoplastic conditions:							
Carcinoma of head of pancreas with obstruction	2	2	—	2	—	—	50–152
Secondary carcinoma (bronchus, stomach, pancreas)	3	3	—	3	—	—	43–388
Miscellaneous conditions:							
Appendicitis	1	—	—	—	—	1	12
Peritoneal abscess	1	—	—	—	—	1	34

With treatment by posture and antacids, the patient is now free of pain.

Of the remaining cases of heart disease in which the estimation was performed, slight elevation of the S.G.O-T. level was found in one case of myocarditis.

CASE IV.—A housewife, aged 54 years, was admitted to hospital with symptoms and signs of congestive heart failure. These had been present for nine months and had proved resistant to treatment. The heart was considerably enlarged and she had a left hydrothorax. An electrocardiagram showed atrial fibrillation and left bundle branch block; the S.G.O-T. level was 49 units. Despite treatment with cardiac glycosides and diuretics her condition grew worse. At autopsy (Professor R. E. J. ten Seldam), widespread fibrosis of the myocardium and changes suggestive of active myocarditis of unknown ætiology were seen.

Patients with Hepatic Disease.

The results obtained in cases of hepatic disease are shown in Table III.

In all cases in which the occurrence of polygonal cell necrosis was suspected on clinical grounds, or was proved histologically, S.G.O-T. levels were elevated. In one case of cirrhosis in which a normal level was encountered, it is reasonable to suppose that this pathological process was inactive. In two cases of intraabdominal suppurative disease (appendicitis and peritoneal abscess), it was suspected that portal pyæmia was a complication. However, the G.O-T. values were normal, and at subsequent laparotomy extension of inflammation beyond the local inflammatory site had not occurred.

CASE V.—A motor-truck driver, aged 40 years, who had consumed two to three bottles of wine a day for many years, was admitted to hospital with symptoms of peripheral neuritis. The liver was found to be considerably enlarged. The only abnormality found on biochemical tests of liver function was elevation of the bilirubin level in the serum (2·5 milligrammes per 100 millilitres). The S.G.O-T. levels on three occasions were found to be 216, 362 and 198 units respectively. Liver biopsy showed increased fatty infiltration with early diffuse fibrosis. An occasional parenchymal cell appeared necrotic. After 26 days of therapy, including a protein-rich diet with vitamin supplements, the patient was discharged from hospital.

DISCUSSION.

It has been confirmed that, when the estimation is made within 24 to 48 hours after the onset of pain, the finding of elevated S.G.O-T. levels is helpful in the diagnosis of myocardial infarction. The clinician is often faced with several diagnostic difficulties. A history of pain may be suggestive of myocardial infarction, but electrocardiographic confirmation may be lacking; either *Q*, *S-T* and *T* wave changes may not be visible, or the picture may be obscured by bundle branch block, old infarction or Wolff-Parkinson-White syndrome.

In these circumstances, the finding of elevated S.G.O-T. values is most helpful; this may precede changes in the erythrocyte sedimentation rate and leucocyte count, which in any case may be of inconstant occurrence and significance. In other cases, difficulty may be experienced in differentiating on clinical grounds between acute coronary insufficiency and infarction. Our experience confirms that of other workers—that ischæmia without infarction is not associated with a rise in S.G.O-T. levels.

Hiatus hernia, acute benign pericarditis and dissecting aneurysm of the aorta are often difficult to distinguish on both clinical and electrocardiographic grounds from myocardial infarction. In these circumstances, S.G.O-T. estimation may also be of vital importance. Indeed, in the definitive diagnosis of myocardial infarction, S.G.O-T. estimation must rank as one of the most helpful aids to the clinician to appear since the introduction of unipolar electrocardiography.

In hepatic disease S.G.O-T. levels would appear to be a most sensitive index of parenchymal damage, so sensitive that serial study may prove to be of help in assessing progress and prognosis in cases of parenchymal liver disease. S.G.O-T. estimations are not likely to replace the conventional battery of tests of liver function used to differentiate between jaundice due to obstruction and that due to parenchymal disease, but they supply useful supplementary information in cases in which conventional biochemical data are unhelpful.

SUMMARY AND CONCLUSIONS.

1. Recent work on transaminase enzyme systems and the history of the application of estimation of these enzymes in serum to clinical problems has been briefly reviewed.

2. Serum glutamic-oxalacetic transaminase activity has been determined in 60 patients suffering from a variety of cardiac and hepatic diseases.

3. The S.G.O-T. level was increased consistently in cases of acute myocardial necrosis within 24 hours of the onset of ischæmic pain.

4. In contradistinction to myocardial infarction, the S.G.O-T. level was not elevated in cases of coronary insufficiency. The importance of S.G.O-T. estimation in differentiating between "insufficiency" and infarction and in differentiating myocardial infarction from other causes of chest pain is discussed.

5. Attention is drawn to the value of the estimation of the S.G.O-T. level in both diagnosis and prognosis of hepatic lesions, as raised levels of the enzyme were consistently found in patients with liver necrosis.

ACKNOWLEDGEMENTS.

We wish to acknowledge the help given us in the preparation of this paper by Dr. D. H. Curnow and Professor E. G. Saint, and to thank the honorary medical staff of the Royal Perth Hospital, who provided the clinical material.

REFERENCES.

AWAPARA, J., and SEALE, B. (1952), "Distribution of Transaminase in Rat Organs", *J. Biol. Chem.*, 194: 497.

CABAUD, P., LEEPER, R., and WROBLEWSKI, F. (1957), "Serum Glutamic Oxalacetic Transaminase", *Am. J. Clin. Path.*, 26: 1101.

CHINSKY, M., SHMAGRANOFF, G. L., and SHERRY, S. (1956), "Serum Transaminase Activity Observations in a Large Group of Patients", *J. Lab. & Clin. Med.*, 47: 108.

DE RITIS, F., COLTORTI, M., and GIUSTI, G. (1955), "*Attività transaminasiche del siero nell 'epatite virale*", *Ital. Soc. Exp. Biol.*, 31: 894.

KARMEN, A. (1955), "A Note on the Spectrophotometric Assay of Glutamic Oxalacetic Transaminase in Human Blood Serum", *J. Clin. Investigation*, 34: 131 (Appendix).

KARMEN, A., WROBLEWSKI, F., and LA DUE, J. S. (1955), "Transaminase Activity in Human Blood", *J. Clin. Investigation*, 34: 126.

KRAUSE, S., and KRAUSE, G. (1956), "Serum Glutamic Oxalacetic Aminopherase (Transaminase) Determinations. Value in the Diagnosis of Acute Myocardial Infarction in the Presence of Left Bundle Branch Block", *J.A.M.A.*, 161: 144.

LA DUE, J. S., WROBLEWSKI, F., and KARMEN, A. (1954), "Serum Glutamic Oxalacetic Transaminase Activity in Human Transmural Myocardial Infarction", *Science*, 120: 497.

MASON, J. H., and WROBLEWSKI, F. (1957), "Serum Glutamic Oxalacetic Transaminase Activity in Experimental Disease States", *Arch. Int. Med.*, 99: 245.

MOLANDER, D. W., WROBLEWSKI, F., and LA DUE, J. S. (1955), "Serum Glutamic Oxalacetic Transaminase as an Index of Hepatocellular Integrity", *J. Lab. & Clin. Med.*, 46: 331.

NYDICK, I., RUBOSSOGER, P., WROBLEWSKI, F., and LA DUE, J. S. (1957), "Variations in Serum Glutamic Oxalacetic Transaminase Activity in Experimental and Clinical Coronary Insufficiency, Pericarditis and Pulmonary Infarction", *Circulation*, 15: 324.

REITMAN, S., and FRANKEL, S. (1957), "A Colorimetric Method for the Determination of Serum Glutamic Oxalacetic and Glutamic Pyruvic Transaminases", *Am. J. Clin. Path.*, 28: 56.

WROBLEWSKI, F., and CABAUD, P. (1957), "Colorimetric Measurement of Serum Glutamic Pyruvic Transaminase", *Am. J. Clin. Path.*, 27: 235.

WROBLEWSKI, F., and LA DUE, J. A. (1954), "Serum Glutamic Oxalacetic Transaminase as an Index of Liver Destruction and Disease", *J. Lab. & Clin. Med.*, 44: 958.

Reviews.

Modern Medicine for Nurses. By Patria Asher, M.D., M.R.C.P., with a chapter on mental ill health by Portia Holman, M.D., M.R.C.P., D.P.M.; Fourth Edition; 1957. London, Toronto, Melbourne, Cape Town: William Heinemann (Medical Books), Limited. 8½" × 5½", pp. 392, with 99 illustrations. Price: 22s. 6d.

THIS is an excellent book. It is compact and comprehensive, yet clearly, simply and concisely written, so that it is easy and interesting to read and understand. It is well within the scope of the trainee nurse, and the price is reasonable. Some chapters, such as that on "Rest", could be profitably read by trained nurses as well as those in training.

Kinetics and Thermodynamics in Biochemistry. By H. Geoffrey Bray, D.Sc. (Birm.), and Kenneth White, B.Sc. (Lond.), Ph.D. (Birm.), F.R.I.C.; 1957. London: J. and A. Churchill, Limited. 9¼" × 6", pp. 356, with 71 illustrations. Price: 42s.

IT becomes increasingly apparent that many problems which confront the biochemist are essentially of a physicochemical nature, and this book is a most interesting and timely publication. The text will be rewarding reading for the biologist interested in the theory of kinetics and thermodynamics, and in the application of these disciplines to biochemical and biological problems. The book should be of interest also to "physical chemists who will find familiar ideas applied to unfamiliar problems".

So that the book may be comprehensible to as many types of readers as possible, each main topic is introduced at an elementary level, and the authors have deliberately kept the mathematical treatments as simple as possible. The treatment of the application of kinetics and thermodynamics proceeds from the simplest chemical reactions through complex biochemical reaction networks to the whole organism. Topics discussed include structure and biochemical function, energetic coupling and active transport, equilibria concerning oxidation-reduction reactions and phosphate compounds, the kinetics of open reaction systems and of isotopic reactions, a logical rather than historical development of enzyme kinetics, pathways of free energy, rate determining processes and control of metabolism.

Dermatologic Formulary: From the New York Skin Cancer Unit, Service of Dermatology (Dr. Marion B. Sulzberger, Director). Edited by Frances Pascher, M.D.; revised 1957. New York: Paul B. Hoeber. 7½" × 5", pp. 191. Price: $4.00.

THE second edition of "Dermatologic Formulary" maintains the high standard set by the first edition.

Therapeutic advances of the last few years are incorporated, especially in the field of corticosteroids and antibiotics. Amongst the new antibiotics is "Nystatin" (tablets for oral administration). A new feature is that the various formulæ are listed under the sections "Topical Remedies" and "Systemic Therapy". The latter is divided into (*a*) medicaments for oral use and (*b*) medicaments for parenteral use. Actions, uses, indications, contraindications and directions are still mentioned under each item, as in the first edition.

This book can be thoroughly recommended, not only to the practising dermatologist, but also to the general practitioner who is interested in dermatology.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Introduction to Biostatistics", by Huidah Bancroft, Ph.D.; 1957. New York: Paul B. Hoeber. 9¼" × 6", pp. 224, with 37 illustrations. Price: $5.75.

This is "the third revision of a series of mimeographed notes prepared for use in the teaching of biostatistics to sophomore medical students at Tulane University".

"Combined Textbook of Obstetrics and Gynæcology: For Students and Practitioners", edited by Dugald Baird, B.Sc., M.D., D.P.H., F.R.C.O.G.; Sixth Edition; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 9¼" × 6¼", pp. 948, with 492 illustrations. Price: 95s. (English).

Extensive revision has been made since the previous edition was published in 1950. The format has been changed as well as the text.

"The Diagnosis and Treatment of Infections", by D. Geraint James, M.A., M.D. (Cantab.), M.R.C.P. (Lond.); 1957. Oxford: Blackwell Scientific Publications. 8¼" × 5½", pp. 244, with illustrations. Price: 30s. (English).

This book aims to synthesize the clinical and the laboratory aspects of infections for the student and practitioner, and for the mutual benefit of clinician and laboratory worker.

"The Malabsorption Syndrome", a Mount Sinai Hospital monograph, edited by David Adlersberg; 1957. New York: Grune and Stratton. 9¼" × 7", pp. 256, with many illustrations. Price not stated.

A detailed consideration of the conditions known variously as idiopathic steatorrhœa or non-tropical sprue, tropical sprue and cœliac disease.

"Advances in Tuberculosis Research. Volume 8: BCG—A Discussion of its Use and Application", edited by Dr. Hans Birkhäuser and Dr. Hubert Bloch; 1957. Oxford: Blackwell Scientific Publications. 9¼" × 6¼", pp. 328, with five illustrations. Price: 60s. (English).

The volume attempts to emphasize the controversial questions concerning BCG.

"World Directory of Medical Schools"; 1957. Geneva: World Health Organization. 9¼" × 6¼", pp. 314. Price (cloth-bound edition): £1 5s., $5.00, Sw. fr. 15.

A list of institutions of medical education in more than 80 countries, with a few pertinent facts about each and some general statements describing the salient features of undergraduate medical training in each country.

The Medical Journal of Australia

SATURDAY, JANUARY 18, 1958.

THE MURRAY REPORT.

PROFESSOR C. R. B. BLACKBURN, in an article on "The University and Clinical Medicine" in the University of Sydney *Gazette* of April, 1957, wrote:

> There are immediate and future difficulties in regard to numbers and finance, the plenty and the poverty, but they cannot override the necessity for the production of a working plan for now and the future.

This statement related to the training of medical students in the University of Sydney, but it did in fact sum up the overall situation throughout all Australian universities—"the plenty and the poverty" presenting a seemingly insoluble dilemma. Now, with the nearest thing to the waving of a magic wand that we might ever expect to see, the solution appears to be in sight. The Report of the Committee on Australian Universities, under the chairmanship of Sir Keith Murray, has been made, its principal recommendations have been accepted by the Commonwealth Government, and practical steps are already being taken to put them into effect. The Report is a notable document, which should be widely read, not so much for the sane and practical way in which it deals with immediate problems, but for the excellent picture it gives of the place of the university in the Australian community and the vision it stimulates for the future.

The terms of reference for the committee were that it should indicate ways in which the universities might be organized so as to ensure that their long-term pattern of development is in the best interests of the nation, and in particular to inquire into such matters as (i) the role of the university in the Australian community, (ii) the extension and coordination of university facilities, (iii) technological education at university level, and (iv) the financial needs of universities and appropriate means of providing for these needs. After an extensive survey of all the Australian universities, carried out with commendable thoroughness in a short time, the members of the committee state candidly: "We had hoped to find that they [the Australian universities] were at present adequately staffed and equipped to discharge their heavy responsibilities to the students and to the nation. This is, unfortunately, far from the case." This finding will have occasioned no surprise to the universities, and probably little to the Commonwealth Government, but the point is that the committee indicates how the situation may be remedied. It will cost a great deal of money, but the Prime Minister has promised that this will be made available.

We cannot attempt even to summarize the details of the report, but it is made clear that the universities must be enabled to carry out effectively their twin roles of education and research. The report emphasizes the need for more, not less, graduates in Australia. The high failure rate in our universities is described as "a national extravagance which can ill be afforded". The answer is not to raise university entrance standards and in other ways to restrict graduation to the outstanding few, but to ensure that all the large number of students who can benefit by a university education shall be given the maximum opportunity to enter universities and to graduate successfully. The "poverty" needs to be overcome and advantage taken of the "plenty" of Professor Blackburn's phrase. At the same time steps should be taken to overcome the "general weakness of honours work and of post-graduate training and research work" which is described as "also a disquieting feature of the Australian universities". Recommendations are made also on the questions of accommodation, equipment and staff salaries, on the future role of the universities particularly in relation to increasing demands for technical training, on problems of expansion and administration and on the need for an Australian University Grants Committee. Specific financial recommendations are made to meet the immediate situation. That the Commonwealth Government has responded so fully is most gratifying and amply justifies the Prime Minister's comment, when he dealt with the Report in the House of Representatives on the evening of November 28, 1957, that it was "rather a special night" in his political life. It would be a considerable understatement to say that it was "rather a special night" in the university life of Australia.

Two matters of specifically medical interest in the Report require separate comment. The first is of relatively minor importance, but warrants careful thought—namely, the need for student health schemes. Despite favourable reports on such schemes in universities overseas, little has been done in the matter in Australia. The committee wisely does not think that a university medical officer should, in general, undertake the treatment of sick students, but sees a useful role for him as an adviser to students and to university teaching staffs.

The second specifically medical matter is of far-reaching importance and relates to the foundation of new medical schools. The Report refers in general terms to the situation of the University of Melbourne, which is not regarded as capable of a great deal of further expansion, and points out that it has submitted a separate report to the Victorian State Government. However, it is indicated that this deals with the establishment of a second university in Melbourne, which is likely to have its own medical school. Further details of this proposal will be awaited with much interest. There will be some disappointment no doubt at the committee's statement that it cannot support the proposal for a Faculty of Medicine in the University of Tasmania, but the practical difficulties are probably too great at present. The committee favours the establishment of a second medical school in New South Wales, and active steps are already being taken to bring this about. As it is a major matter, we shall refer to it in a separate article. The important point is that both Sydney and Melbourne should each have a new medical school within the reasonably near future. Following so closely on the establishment of the Western Australian Medical School, this

should help to overcome the present intolerable congestion in the earlier years of the older medical schools, and, with better facilities for teaching and more encouraging opportunities for the students, reduce the present depressing failure rate. How the increased numbers will be accommodated for clinical teaching will have to be determined, but should not present great difficulty. It is certainly a relief to see light appearing on a hitherto gloomy scene.

Current Comment.

PRIMARY LYMPHŒDEMA.

SINCE 1954, J. B. Kinmonth has published a series of papers on his ingenious technique for visualizing the peripheral lymph vessels. The theory of this technique is simple enough: a suitable dye is injected intravenously near the extremity of a limb; the dye becomes concentrated in the lymph channels, making it possible to see the superficial lymph vessels through the skin at certain sites, notably across the middle of the dorsum of the foot; a radioopaque solution is then injected into a lymph-trunk previously made visible by the dye. Owing to the free anastomoses within the network of lymph channels, it is thus possible to obtain a radiographic picture of the entire lymph drainage of a given area.

In their latest paper Kinmonth and his co-workers[1] have applied this technique to the study of 107 cases of primary lymphœdema affecting the lower limbs. In 12 of these the condition was congenital, and in the majority of the remainder it developed before the age of 35 years. Over 70% of all cases were in females. Eighteen of the patients gave a history suggesting a familial incidence. In two of these the condition was in the congenital group and could therefore be classed as an example of Milroy's disease, which the authors regard as being merely a special example of the condition. One or both lower limbs may be affected in about equal proportions, and in a small group some other part of the body was affected as well. In 14 of the patients there was some other congenital anomaly, but seven of these were blood-vascular anomalies, sometimes of a trivial nature.

The results of the lymphangiographic studies showed that in over 50% there was hypoplasia of the lymph-trunks—that is, the lymph-trunks were found to be deficient in size or number or both. A common anomaly was to find only a single lymph-trunk extending up to the groin. A smaller group showed complete aplasia, without any demonstrable subcutaneous lymph-trunks; this was a frequent finding in the more severe grades of the condition. About 25% had varicose lymph-trunks, which were broader than normal and tortuous, and in which valvular incompetence could be demonstrated. The remaining 6% showed dermal backflow alone—that is, backflow from deeper trunks into the capillary network of the skin. The authors regard all these states as being primarily congenital in origin. In some cases the œdema is present at birth, in others it develops later in life. Among the latter, the onset of œdema is sometimes related to some traumatic or inflammatory episode. It is suggested that in these cases the poorly developed lymph vessels had previously been able to maintain drainage, but were inadequate to deal with the added load and were never subsequently able to drain off the excess fluid. The role of inflammation as a possible primary cause of the œdema was carefully considered, and all patients were questioned on this point. In 26 cases there was a history of attacks of inflammation, but in all but two there was evidence that the œdema antedated the infection, and the authors conclude that in such cases the inflammation is secondary, œdema predisposing to infection.

Capillary defect allowing excess formation of fluid has also been suggested as a cause of lymphœdema, but the authors adduce evidence that there is no such defect. They believe that the chief role of the lymphatics as regards drainage is to carry off from the tissue spaces substances of large molecular size, such as would hinder their reabsorption through the walls of the blood capillaries. Failure of this drainage system results in an accumulation of proteins in the tissue spaces, with consequent water retention and œdema.

[1] *Brit. J. Surg.*, 1957, 45: 1 (July).

FLUORIDE DENTIFRICE AND STOMATITIS.

THE use of fluoride in drinking water to lessen the incidence of dental caries is widespread. Where this is not adopted, it has been recommended that fluorides be applied locally to the gums, and commercial dentifrices containing fluorides are available. In this regard it has been recognized that local application might be injurious to the soft tissues. T. E. Douglas[1] has reported on 133 patients with stomatitis. There was nothing specific in the appearance of the lesions. Shallow, superficially ulcerated areas, usually with a whitish exudate on the surface, were seen particularly on the buccal mucosa opposite the teeth. The tongue, hard palate, soft palate, floor of the mouth, gingival region and oral part of the pharynx might also show such lesions. The breath was usually somewhat fœtid. The common factor in all these cases was the use of a dental powder or cream containing a fluoride. Thirty-two of the patients cooperated over a range of two to six courses of three weeks of the dentifrice containing fluoride and three weeks of a dentifrice that did not contain fluoride. In each case the stomatitis appeared during the period of the fluoride-containing dentifrice and disappeared in the fluoride-free period, although the disappearance of the lesions took longer with increasing numbers of courses.

It appears that treatment of the stomatitis is simple in the early stages—it merely involves changing to a dentifrice containing no fluorides. When the lesions are more advanced, painting with 10% to 20% silver nitrate solution is helpful. After long-continued use of the fluoride-containing dentifrice, healing of the stomatitis was found to be difficult and slow. Antibiotics had little or no effect. One might expect similar stomatitis to develop when solutions of fluorides are rubbed on the gums, a procedure recommended by some dentists. Diagnosis of the condition is easy if one thinks of fluoride therapy and obtains a history of its use.

[1] *Northwest Med.*, 1957, 25: 1037 (September).

THE SURGERY OF PORTAL OBSTRUCTION.

IT is just eighty years since Eck, a Russian physiologist, first published his account of an operation to establish a fistula between the portal vein and the inferior *vena cava*. His own object was experimental research with animals, but he suggested that a similar operation might be used for the relief of portal obstruction in man. However, it was more than 30 years later before the progress of abdominal surgery reached a stage where it was possible to attempt this with a reasonable hope of success. Several surgeons attempted the operation about 1910, but in spite of one or two brilliant successes the mortality was prohibitive, and the operation was for the time being abandoned. Subsequently the operation of omentopexy gained favour for a time, but this was not very satisfactory. Then, in 1945, Blakemore and Whipple in America pioneered the modern operations of porta-caval and spleno-renal shunt. These operations are now well established, and are already the subject of a considerable literature. A year ago Professor R. M. Walker,[2] of Bristol, reviewed a series of 56 patients who had had end-to-side porta-caval anastomoses performed to relieve symptoms of portal hypertension due to intrahepatic obstruction. He

[2] *Lancet*, 1957, 1: 57 (January 12).

has made routine use of trans-splenic venography to localize the venous obstruction beforehand, and this is clearly most important in the selection of patients for operation. The ages of patients at the time of operation ranged from seven to 57 years. In this series there were only three post-operative deaths, all from hepatic coma. The most important complication was the neuropathy which is a known risk in portal systemic shunts; in addition to the three patients who died, nine others had episodes of encephalopathy, but most of these responded quickly to dietetic treatment. Professor Walker regards this complication as rare in those whose liver function is normal. Two patients who had had previous splenectomies had a recurrence of hæmorrhage after operation, as did one other who had had a previous spleno-renal shunt performed; but none of the remaining 50 had any further bleeding up to the time of his review.

Three other recent papers review results obtained at some American centres. Professor C. S. Welch and A. G. Ramos[1] in Albany, New York, have given their results in 40 consecutive cases. They experienced an operative mortality of 15%, but had only two deaths in their last 20 cases. They advocate hypotensive spinal anæsthesia, stating that this reduces blood loss and operating time, and has no deleterious effects. The long-term survival rate for their patients was 58%, and they suggest that if greater benefit is to be obtained, the operation should be performed on selected patients before œsophageal bleeding occurs. There was a recurrence of bleeding after operation in 20% of their patients, most of these being patients who had had a spleno-renal shunt performed.

In Chicago, W. S. Dye and co-workers[2] have reported on 77 cases in which 47 spleno-renal shunts and 22 porta-caval shunts were effected (eight other patients were subjected to multiple procedures). Of the 47 who had a spleno-renal shunt, 26 survived at the time when the paper was written (with a minimum period of six months' survival), and of the 22 who had a porta-caval shunt, 14 survived. Post-shunt bleeding occurred in 36% of those who had a porta-caval shunt, and in 21% of those who had a spleno-renal shunt. These authors do not consider that surgery is justified in patients who have never had a hæmorrhage. They believe that abstinence from alcohol is a most important factor, and think that many of the poor results in surgically treated patients are due to continued drinking of alcohol.

G. A. Hallenbeck and E. Shocket,[3] from Rochester, Minnesota, discuss their results in 55 cases in which spleno-renal or porta-caval shunts were performed between 1951 and 1955. Forty-seven of the patients had intrahepatic portal obstruction, and eight had extrahepatic obstruction. Of the former, 44 had previously experienced hæmatemesis. Of those with intrahepatic obstruction, 29 underwent operation to establish a porta-caval shunt, and 18 to have a spleno-renal shunt. There were seven post-operative deaths among the former, and only one among the latter; but the authors point out that most of their poor risk patients were in the former group, as they consider that the porta-caval shunt operation is more effective in relieving portal hypertension. Of 23 patients who survived the porta-caval shunt operation, five subsequently had hæmorrhages, and of 18 who survived the spleno-renal shunt operation, four subsequently had hæmorrhages. Two of these nine patients died soon after the hæmorrhage. These 47 patients experienced a total of 75 hæmorrhages during the 12 months before anastomoses were performed, and the surviving patients had only four hæmorrhages during the 12 months following operation. Thus, although large, well-placed porta-caval or spleno-renal shunts do not provide absolute protection against œsophageal bleeding, they are effective in most instances. When splenic portagrams indicate that the splenic vein is large, these authors are inclined to use spleno-renal anastomosis because of the development of serious neurological symptoms, which followed porta-caval anastomosis in three of their cases. Of their eight patients with extra-hepatic portal obstruction, four were children aged four to six years, and in these the results of spleno-renal shunt operations were unsatisfactory, because of the small calibre of the splenic veins. In such cases the authors advise transœsophageal ligation of varices, hoping to obtain remission from bleeding till the children are older.

In another paper, Professor Welch[1] discusses the management of patients with massive hæmorrhage from œsophageal varices due to liver cirrhosis. He points out that these patients are suffering from forced feeding of easily absorbable protein (blood), with which an already diseased liver is unable to cope. In such patients there may be a progressive rise in blood ammonia level, leading to coma and death. (In patients with normal liver function who are subjects of massive gastro-intestinal hæmorrhage from other causes, ammonia is rapidly removed from the circulation.) Consequently, in these cases control of hæmorrhage has a double urgency; it is not sufficient merely to replace lost blood. Professor Welch advocates emergency ligation of the œsophageal varices, to be followed later by a porta-caval shunt, as ligation alone gives only a temporary respite. For immediate control of bleeding he uses a Sengstaken-Blakemore tamponade tube. Blood lost is replaced by transfusion, and the gastro-intestinal tract is emptied by means of hourly doses of milk of magnesia administered through the gastric tube accompanied by repeated enemata. Ligation is then performed 24 to 72 hours after the patient's admission to hospital. Later on, when the patient is stabilized, one or other form of porta-caval anastomosis is effected.

The differences among these series are probably to a considerable extent a matter of case selection. Walker attributes his good results to careful selection of cases; and those who accept poor-risk patients inevitably incur higher mortality rates. Clearly there are still considerable divergencies of opinion both on matters of technique and on the precise indications for operation.

[1] *Surgery*, 1957, 41:756 (May).
[2] *Arch. Surg.*, 1957, 74:959 (June).
[3] *Surg., Gynec. & Obst.*, 1957, 105:49 (July).

ABSTRACTS OF SOVIET MEDICINE.

THE Excerpta Medica Foundation was established ten years ago with the object of providing a comprehensive medical abstracting service in the English language. Their latest venture, *Abstracts of Soviet Medicine*,[2] is intended as a quarterly publication in two parts, dealing respectively with the basic medical sciences (Part A) and clinical medicine (Part B). The first number of Part A consists of abstracts of almost 250 papers arranged in five major groups: (i) anatomy, embryology and histology, (ii) physiology, biochemistry and pharmacology, (iii) endocrinology, (iv) medical microbiology, immunology and serology, (v) general pathology and pathological anatomy. Of these sections that devoted to endocrinology is the longest. Among the topics which catch the eye on a quick perusal of the publication are: a description of a 14-day-old human embryo; an account of experiments on the human heart after death from various causes (in one case the activity of atria and ventricles of the heart of a premature infant was started again 99 hours after death); the resuscitation of puppies 10 minutes after clinical death from exsanguination, without loss of conditioned reflexes established before death; a suggestion that the incidence of skin cancer, as well as being influenced by climatic factors, bears an inverse ratio to the magnesium content of the soil. The material for these abstracts is provided by a panel of members of the U.S.S.R. Academy of Medical Sciences. As would be expected, they record a substantial volume of original research on a wide variety of topics, and this publication should be of great assistance to anyone interested in the progress of Russian research in his own particular field.

[1] *Surgery*, 1957, 41:1029 (June) (see also W. J. McDermott *et alii*, *Ann. Surg.*, September, 1956, abstracted in M. J. AUSTRALIA, 1957, 1:327).

[2] *Abstracts of Soviet Medicine*, Part A, Basic Medical Sciences, Volume 1, Number 1, 1957. Amsterdam and New York: Excerpta Medica Foundation. 9¾" × 6½", pp. 102. Annual subscription price: $15.00.

Abstracts from Medical Literature.

PÆDIATRICS.

Œsophageal Stricture from "Clinitest" Tablets.

J. P. CANBY (*J. Pediat.*, January, 1957) describes the development of œsophageal stricture in two children who swallowed "Clinitest" tablets. These tablets consist of copper sulphate, caustic soda, sodium bicarbonate and citric acid. The first child, aged 3 years, swallowed an unknown number of tablets which caused immediate coughing and choking. He was given water to drink at once and taken to the local hospital where gastric lavage was performed. During the next few days his appetite declined and three weeks later he began vomiting. Six weeks after the ingestion he refused to drink even clear liquids, and a barium bolus and œsophagoscopy revealed almost complete obstruction of the œsophagus by a stricture at the level of the third thoracic vertebra. The second child, aged 23 months, swallowed one "Clinitest" tablet and apparently vomited part of it. A week later he began to vomit, and after five weeks even had difficulty in swallowing liquids. Examination with a barium bolus revealed a localized constriction between the upper and middle thirds of the œsophagus.

Patent Ductus Arteriosus.

L. M. HOLVE and F. H. ADAMS (*J. Pediat.*, August, 1957) emphasize the importance of recognizing the presence of a patent *ductus arteriosus* with a large left-to-right shunt in infants who lack the classical machinery murmur usually seen in older children. This classical murmur is sometimes also absent in the older child in whom a large shunt is present. The usual findings in these infants are a failure to grow and develop normally, tachypnœa, dyspnœa, repeated respiratory infections and easy fatigability. A wide pulse pressure is often present. Usually there is a harsh systolic murmur along the left sternal border and commonly there is a short diastolic component, though this is not always present. Cardiac enlargement of varying degrees is present. The electrocardiogram shows no consistent pattern. The diagnosis can be confirmed with certainty by means of cardiac catheterization or retrograde aortography. Operation is usually possible with very satisfactory results.

Generalized Cytomegalic Inclusion Disease.

A. E. MCELDRESH and J. B. ABEY (*J. Pediat.*, August, 1957) describe in detail three cases of generalized cytomegalic inclusion disease in infants in which the diagnosis was confirmed by the finding of inclusion-bearing cells in the urine. The infants presented with petechiæ and purpuric spots on the first day of life, and soon developed jaundice and enlargement of the liver and spleen. In the first child progress was unsatisfactory. Petechiæ kept recurring, hepato-splenomegaly persisted, there was irregular fever, evidence of mental retardation soon became apparent, the anterior fontanelle became tense, the neck rigid and a generalized tremor developed. The spinal fluid contained a few lymphocytes and increased protein, and smears of the fluid obtained by subdural tap contained a number of cells with large deep purple intranuclear inclusion bodies when stained by hæmatoxylin and eosin. Autopsy revealed hydrocephalus and anencephaly with cerebral calcification and the presence of inclusion bodies in scattered viscera. Focal infiltrations of lymphocytes in a number of viscera were noted and were thought to represent reaction to preexisting inclusion-bearing cells. The second infant progressed more satisfactorily. He had cyanosis and scattered petechiæ at birth. Jaundice occurred at 12 hours of age and the petechiæ extended to the trunk and extremities. He made satisfactory early progress, but the jaundice persisted and enlargement of the spleen and liver became apparent. At seven weeks of age he began to have irregular fever. Smears of the urinary sediment revealed the characteristic cells of cytomegalic inclusion disease. As he developed, some mental retardation became apparent, but with considerable scatter. At five months he held up his head and was able to roll over, but he did not smile until five months of age. He sat alone at the age of 11 months and at 19 months walked well with support and spoke a few words. At 26 months of age he had a good vocabulary, used simple sentences, built a tower of five blocks and walked fairly well, but with a wide-based slightly ataxic gait. Enlargement of the liver and spleen was still present. The third child had petechiæ and enlargement of the spleen at birth and jaundice developed in a few hours. On the third day of his life the diagnosis of inclusion body disease was established by the demonstration of characteristic cells in the urine. The child failed to gain normally for the first month of life and had recurrent petechiæ. After this progress was satisfactory, but there is now some evidence of a little retardation. The child did not sit alone until nine months of age but walked at 15 months. The development of speech was slow and at the age of 20 months he had only three words. At the time of reporting he was 35 months of age and his mental progress had been satisfactory except for considerable retardation in speech and some slight delay in motor activities.

Long Term Evaluation of the Juvenile Diabetic.

T. H. JOOS and J. A. JOHNSTON (*J. Pediat.*, February, 1957) outline their experience with a group of 150 cases diagnosed under the age of 15 years and followed for periods of from one to 30 years. They give special attention to 44 patients who have been followed for more than 10 years. The patients are for the most part middle class and have intelligent parents. In this group of 44 patients 26 or 59% had some vascular complication. The average time of the onset of the first evidence of any complication was 11 to 12 years after the initial diagnosis of diabetes had been made. The severity of the complication varied greatly. Three patients died of Kimmelstiel-Wilson disease, another had bilateral cataracts and very extensive retinal hæmorrhages. In the total of 150 patients there had been 10 deaths, six in coma, three cases of Kimmelstiel-Wilson disease and one of tuberculosis. The most common complications were changes in retinal vessels and punctate retinal hæmorrhages. The authors believe that the better the control of the diabetes, the fewer are the complications. They favour a controlled dietary regime and believe that the controversy between this and a free diet is swinging in favour of control. They point out that caloric and insulin requirements in the young diabetic are not stable. Each shows a steady increase until after puberty. They admit the difficulties of an extremely rigid programme and suggest that one which provides a moderate amount of carbohydrate and allows a 2+ Benedict's reaction once a day to guard against insulin reaction, affords a good middle ground to follow.

Clotting Factors in the Newborn.

J. W. FRESH *et alii* (*Pediatrics*, February, 1957) have carried out an investigation of the levels of prothrombin, proconvertin and proaccelerin in the blood of normal infants and discuss its relationship to vitamin *K*. They carried out investigations on the concentration of these factors in the blood of 114 newborn infants for the first six weeks of life. It was found that prothrombin and proconvertin (factor 7) were low at birth and fell still lower in the next two or three days, then gradually and steadily rose. Proaccelerin (factor 5) on the other hand was usually above normal at birth. These fluctuations in prothrombin and proconvertin were not significantly affected by small doses of vitamin *K* given to the mother before delivery or to the infant at birth. The authors suggest that it is the excess of proaccelerin which compensates for the deficiency in the other two factors and prevents the vast majority of newborn infants from developing any bleeding condition. They do not discuss whether it is a deficiency of prothrombin or of proconvertin that is responsible for hæmorrhagic disease of the newborn, nor do they make any recommendations or comments about the comparative value of vitamin *K* or of vitamin K_1 oxide in the treatment of this condition. They believe that there is no advantage in giving a single dose of vitamin *K* to the mother during pregnancy or to the infant at birth; but they are carrying out further studies to see whether vitamin *K* supplements given for a longer time during pregnancy will have any effect in preventing the deficiency of these clotting factors in the newborn.

Facial Paralysis in Children.

R. S. PAINE (*Pediatrics*, February, 1957) discusses the anatomy of the facial nerve and the very many etiological factors that can cause congenital and acquired facial paralysis in children. The differential diagnosis between the more important of these is discussed, and an attempt made to evaluate the possible methods of treatment that are available. The difficulty of this evaluation lies in the high recovery rate without treatment,

which it is believed approximates to 90% in children. Physical therapy by means of local application of heat, electrical stimulation, massage, splinting of the face and active exercises in front of a mirror is discussed and varying opinions about the value of these procedures are quoted. The wisdom of placing a drop of mineral oil or olive oil in the affected eye at night to prevent drying is emphasized. Surgical decompression of the facial canal is usually reserved for cases in which there is no evidence of recovery after the first few weeks. The author believes that if this procedure is to be of value it should be done before degeneration has taken place, that is, within eighteen days of the onset of paralysis. He considers that there is considerable justification for attempting decompression of the facial canal in patients in whom there is no apparent recovery and a beginning reaction of degeneration fourteen days after the onset of the disease. The place of cortisone is discussed in detail, but evaluation is again difficult because of the high recovery rate in untreated patients. If a careful study of a case of facial paralysis indicates that it is of idiopathic origin, then prompt treatment with cortisone should be used. It is claimed that the average recovery time is favourably influenced, at least in adults and probably also in children. The author suggests that the use of cortisone may eliminate the need for consideration of surgical decompression of the facial canal. Before it is used, care should be taken to exclude the presence of *otitis media* and poliomyelitis. The treatment of persisting facial paralysis by means of nerve grafts, plastic surgical procedures of the face and the use of crocodile tears is briefly discussed.

Interstitial Plasma Cell Pneumonia.

D. C. GAJDUSEK (*Pediatrics*, April, 1957) reviews interstitial plasma cell pneumonia of premature and young infants and the aetiological agent, *Pneumocystis carinii*. This disease mainly affects infants and especially premature infants. The disease is usually of slow, insidious onset, with restlessness, languor and poor feeding. Tachypnœa and a peculiar bluish cyanosis about the mouth and under the eyes may occur. The lung fields may be free of physical signs, cough is usually not prominent, there may be no coryza, no fever and no weight loss, yet the respiratory rate continues to increase and respiration may become largely abdominal. The fully developed clinical picture appears after one to two weeks of illness, with dyspnœa, dilatation of the *alæ nasi*, sternal retraction, pronounced use of the auxiliary muscles of respiration, cyanosis and marked increase in respiratory rate. There is a bluish-white facial pallor and an anxious expression, and sudden, exhausting attacks of coughing sometimes occur. Physical findings remain minimal, with only fine crepitant rales on coughing or deep breathing and sometimes patchy impairment of resonance and sometimes areas of bronchial breathing. The discrepancy between the severity of dyspnœa and the paucity of physical signs is typical. Interstitial emphysema develops. Death occurs in about 25% of patients, and the others slowly recover. Even in the late stages fever is absent or only slight. The duration is usually four to six weeks, though the insidious onset makes it difficult to judge this accurately. Radiological examination reveals a diffuse, ground-glass cloudiness of the lung with some lighter areas produced by the emphysema. Autopsy reveals pale grey almost airless lungs which are firm, heavy and large, and do not collapse when the chest is opened. There may be extensive mediastinal emphysema or spontaneous pneumothorax. Histological examination reveals a fairly solid, highly cellular appearance, little resembling lung tissue. There is extensive mononuclear cell infiltration, and polymorphs are few or absent. The cells of the mononuclear cell exudate have been called plasma cells and histiocytes, but Swiss workers have recently affirmed that they are monocytes and not plasma cells. The alveolæ are filled with a characteristic exudate, which on staining with hæmatoxylin and eosin, appears to be composed of eosinophilic hyaline-like masses, and faintly granular material. High magnification reveals that this exudate has a peculiar honeycomb-like structure. Coloured with Giemsa stain it is seen to contains masses of parasites. These are *Pneumocystis carinii*. This organism was first found almost half a century ago in the lungs of small mammals and of man in Brazil, and subsequently in the lungs of man and of wild laboratory rodents in Europe. It has been demonstrated with increasing frequency in the lungs of patients dead of the interstitial plasma cell pneumonia of premature and young infants. It has been tentatively classed as a protozoan, but many authors now believe it to be a fungus. It has a very wide geographic distribution in wild an domestic animals of many species, and has now been identified as the cause of interstitial plasma cell pneumonia in infants in many countries, including Australia.

ORTHOPÆDIC SURGERY.

Epiphyseal Arrest.

W. T. GREEN and M. ANDERSON (*J. Bone and Joint Surg.*, July, 1957) in a report from the Massachusetts Infantile Paralysis Clinics and the Department of Orthopædic Surgery of the Harvard Medical School, compare epiphysiodesis and stapling as methods of achieving epiphyseal arrest for the correction of discrepancies in length of the lower extremities. In 1947 the same authors published a paper on epiphyseal arrest by epiphysiodesis, and the present publication records the experience gained since that date. The technique of stapling was introduced in 1949. The results of 475 operations, performed by various surgeons at this centre, are reviewed; of these 383 were epiphysiodeses, and 92 were staplings. Whether epiphysiodesis was performed by bone grafting or by drilling of the epiphyseal plate, good results were obtained with few exceptions. The shortening was well corrected, growth was even and over-correction was rare. One patient had severe tibial bowing because of uneven arrest and refused further surgery, and five other patients required second operations to correct irregular growth. There was one bone infection, which finally healed, with an increase in the discrepancy. Failure to achieve predicted results was attributed to one of four main causes: (i) the expected growth of the opposite limb did not take place; (ii) the operation failed to arrest epiphyseal growth; (iii) difficulties were encountered in judging skeletal age, and variations occurred in the rate of growth in individual children; (iv) the relative size of the patient had to be considered—greater correction is necessary in a patient tall for his age than in a short one. The authors emphasize the great importance of pre-operative evaluation of the amount of further growth to be anticipated. Prediction charts used were based on either the Todd atlas for estimation of skeletal maturity or the Greulich-Pyle atlas, and both were found to be satisfactory. The stapling operation had more complications. Problems of localization of the epiphyseal plate made placement difficult. Sometimes the staples were extruded. They all tended to open. Secondary operations were more common, mainly because staples had to be removed for various reasons. The authors emphasize that stapling was used as an alternative method of epiphyseal arrest, not as a method of temporary inhibition. Stapling has a theoretical advantage in that, if over-correction seems likely, growth may be resumed when the staples are removed, but this resumption does not always take place.

Osteogenic Sarcoma.

M. B. COVENTRY and D. C. DAHLIN (*J. Bone & Joint Surg.*, July, 1957) analyse 430 cases of osteogenic sarcoma selected from a group of some 2000 pathologically verified tumours of bone. The osteogenic sarcomata were the largest group after myelomata. The authors define these as being malignant tumours of bone in which the proliferating cells produce osteoid, and subdivides them into osteoblastic, chondroblastic and fibroblastic types; some were anaplastic. Chondrosarcoma and fibrosarcoma are excluded, as the patients fall into a different age group. The study includes a follow-up of 97% of cases. This showed a five-year survival rate of 20% after definitive treatment. The authors maintain that the prognosis of this condition was regarded with undue pessimism until Coley, in 1950, reported 57 cases with a five-year survival period, and drew attention to the need for careful diagnosis and early effective treatment. The authors favour early surgical ablation without prior irradiation. When surgery is attempted, the first step is to take a biopsy specimen from between two tourniquets; a frozen section is then examined, and a definite diagnosis can usually be made from this. If the diagnosis is proved, amputation is performed, preferably through the bone above the one involved. In the case of the commonest site, namely the lower end of the femur, a high femoral amputation is performed, the upper end of the amputated limb is examined and, if necessary, a further amputation can then be performed. The authors give statistics relating to the types of tumours and their relationship to age, site and survival rate.

Special Article.

REHABILITATION: I. THE ROLE OF THE FAMILY DOCTOR.

DURING the past four years, the word "rehabilitation", cumbersome although it may be, has acquired some real significance in Australia and particularly in New South Wales. Centres for the organized team work practised in this field have been started and are progressing rapidly; but at present the patients are being referred when they have been disabled for long periods. The purpose of this series of articles is to describe the various techniques used, and to explain how the family doctor may avail himself of these services as early as possible after the accident or onset of illness.

Before any of the modern techniques are explained, it is necessary to realize that "rehabilitation" is a product of our modern civilization. All conscientious doctors have had the same aims for their patients through the ages; but the numbers of such people have increased so rapidly, with more efficient medical and surgical care, that it is necessary to devise a special service to deal with this aspect of our practice.

Thirty years ago we were happy to use the motor-cars obtainable at that time, but the manufacturers have so improved our means of transport that few people today would choose to ride in a Model T "Ford" car; so with patient care. We must progress as society progresses, and understand the reasons why these new methods of approach to the patients have become necessary.

Before the advent of antibiotics and modern methods of anæsthesia and surgical techniques, most of those more seriously affected by illness and trauma perished. Those who survived were disabled, but few as grossly as we see now; for example, few children with *spina bifida* survived beyond the first two years of life. Now, at Stoke Mandeville, one sees attractive young men and women, although confined to wheel-chairs, doing productive work and even getting married, physically well and mentally alert, with the will to live. To deal adequately with such serious disabilities, a completely new system has had to be evolved. This system is "rehabilitation".

The family doctor sees the patient first, and unless he knows what is available in rehabilitation centres to relieve him of the burden of these worrying people, he is not aware of their potentialities and the assistance that can be afforded him while keeping him constantly in the picture.

Every undertaking in this world, if it is to succeed, requires planning and a goal; if a human being suddenly finds himself completely dependent with an uncertain future, he loses faith, and as well as suffering from physical disability, he becomes psychologically upset, to add to the problem. If a planned approach is adopted from the earliest hours, preventive measures can be undertaken which will save months of pain and suffering and an enormous amount of expense; for example, the patient who has a cerebral catastrophe, to gain the greatest amount of benefit from rehabilitation, should be evaluated within the first 24 hours of the onset of his hemiplegia. If he is not referred for two or three months, valuable time is lost and the hope of full recovery vanishes. If measures are adopted from the earliest hours, it is possible to gain complete functional recovery in most cases in four months—surely a great advance on the methods of 30 years ago.

How May We Accomplish These Aims?

Evaluation.

Evaluation is the keynote of the service; this must be medical (both physical and mental), social and vocational, with the patient's return to productive work as the goal.

Medical Evaluation.

Medical evaluation requires a careful, detailed history of past and present facts concerning the patient, as well as a thorough physical examination to assess the present state of disability. Very careful observation of the progress in the first days and weeks will determine whether the recovery is to be rapid or prolonged. Prophylactic care should be exercised continually during this period of observation—for example, splinting of the affected joints in acute rheumatoid arthritis or in a patient with hemiplegia, or correct positioning of the stump of an amputee to prevent contractures, which are detrimental at a later date to the successful fitting of a prosthesis.

Social Evaluation.

Social evaluation at the time of the accident or illness not only gives moral support to the patient, but saves time and expense both in the beginning and in the future. In these days of domestic difficulties, few homes are equipped to care for the chronically ill, and few people are available, since many members of all households are wage-earners. This is one of the most difficult problems when there is an acute shortage of hospital beds, as, for example, in New South Wales. The almoner gathers all the details and tries to plan the future for the patient's social and economic welfare. When the victim has suffered an injury at work or in a road accident, it is necessary to know his work history before operation is performed, in order to utilize to the best advantage his limbs for his return, if possible, to his previous employment. For example, one frequently deals with compensation cases in which many months have been spent in meticulous plastic surgery to the fingers of a wharf-labourer, who still has to be retrained for a new occupation. The wharf-labourer presents a very different problem from that of the precision worker, who requires all the skills of his fine movements.

Vocational Evaluation.

Vocational evaluation is intimately intermingled with social evaluation, but goes a step further to discover any latent talents possessed by the patient, when his disability renders him unfit for his previous vocation and new skills have to be acquired. The vocational officer interviews the patient and works with the team continually watching the future for the man. This is a morale-builder, and the whole team approach prevents the difficult psychological state, a combination of apprehension and loss of confidence, which is often found in workers' compensation patients.

The Availability of Rehabilitation Services.

How may these services be made available to the family doctor? In a country such as ours, with a scattered population, it is not economically sound to have large centres in every area; but consultation with skilled and trained people is now available by telephone, or by letter, and the future admission of patients to the existing centres can be arranged early in this way. To the busy general practitioner, such a service has proved a blessing all over the world, and is now being accepted as an integral part of ordinary medical care.

The centres at present available in New South Wales are as follows: the Commonwealth Rehabilitation Centre at Mount Wilga, Hornsby, for pensioners and social service beneficiaries; the Rehabilitation Centre, Royal South Sydney Hospital, Zetland, which is staffed by people with extensive training overseas in these modern methods; the Prince of Wales Rehabilitation Centre at Randwick, a department of the convalescent section of Sydney Hospital, also with highly skilled staff; the remedial kitchen at Royal North Shore Hospital; and a very well-equipped occupational therapy centre at St. Vincent's Hospital geared to rehabilitation techniques for patients of that hospital. Centres are under construction at the Eastern Suburbs Hospital and Royal Prince Alfred Hospital, but are not yet functioning.

Recently it has been my privilege to visit 50 different centres in the United States of America, Canada, Great Britain, Scandinavia and Germany, and I am sure that, thanks to all our Australian medical and ancillary personnel who have studied the techniques of rehabilitation overseas at great inconvenience and expense to themselves, we are now able to offer a service equivalent to anything available in other parts of the world. The best centres have all had small beginnings, and it is universally agreed that for social as well as medical reasons this is the correct approach; the aim should be a case load of about 100 patients per day, the ideal size of a centre.

The support of the family doctor for these centres is essential. In return, they offer him a service which will free him of many of his most worrying problems with help and guidance, while the patient still remains his own.

The family doctor is the most important person in the recognition of crippling disease or accident. He sees the patient "from the cradle to the grave", and is the only person trained for overall observation. He first sees the baby with the congenital heart abnormality, congenital amputation of a limb and other congenital deformities, the child with cerebral palsy. He sees all the early cases of infectious disease, and is usually the first person to recognize the early signs of poliomyelitis. He first sees the largest proportion of industrial and road accidents, as well as accidents in the home. In the older age group, he is the first person called to the patient who has had a coronary

occlusion or a cerebro-vascular accident; in fact, he sees the patient from the beginning to the end of the disease or to the end of his life, as the case may be.

In these days of increasing work and increasing knowledge, it is becoming more and more difficult for everyone to acquire all the techniques necessary to bring many of these human catastrophes to a satisfactory ending. To aid in the fulfilment of every doctor's wish—recovery to the fullest extent compatible with the patient's remaining abilities—rehabilitation centres have been established to assist where the disability needs the attention of a team rather than one person. The object of this series of articles is to acquaint the family doctor with the available facilities, so that we can all work together from the earliest hours.

All over the world, the general practitioner plays an ever-increasing role in these centres, and so derives greater and greater benefit from their establishment. The centres cannot function without him; he is welcome at the original evaluation conferences, he supervises the patient in his home, and he is kept in the picture continually. With the cooperation of general practitioners, this is the atmosphere we are striving to create here.

NAOMI WING,
Sydney.

British Medical Association.

VICTORIAN BRANCH: SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held on May 1, 1957, at the Medical Society Hall, 426 Albert Street, East Melbourne.

Subdural Hygroma.

DR. J. B. CURTIS read a paper entitled "Subdural Hygroma" (see page 69).

SIR JAMES PATERSON ROSS, in opening the discussion, congratulated Dr. Curtis on a clear exposition of a difficult subject. He said that although Trotter's classical description of chronic subdural hæmatoma was well known, he was pleased to hear the reference to the contribution of Adam McConnell, who had worked out those cases clearly and convincingly. He was also glad to hear that Dr. Curtis did not accept the theory of an arachnoid tear. The moral of those cases was that when, after acute head injury, patients failed to do well, a surgeon should be prepared to make burr holes and look for a hæmatoma or a hygroma. If the fluid contained protein, osmosis would lead to an increase in its bulk and, therefore, an increase of intracranial pressure, and he wondered whether infection might be a contributing factor. Was it possible that the infant patients quoted had had a specific infection, and was the Wassermann reaction determined? The extraordinary outcome of that condition in some cases was difficult to explain, and it was disappointing to find that, even with the removal of the fluid, some patients did not improve. Was it possible that multiple collections accounted for the return of symptoms, or had the brain failed to expand? If the latter, could anything be done? Doubtless in some cases the original injury and vascular damage accounted for the failure of brain expansion.

Sir James Paterson Ross said that nowadays he did not see much intracranial surgery, and his colleague McConnell, who was actively engaged in that field, did not see many cases in babies, which was contrary to American experience. It might be thought that those cases were being missed; but post-mortem examination showed that not to be so, and he would be interested if Dr. Curtis could throw light on the difference between English and American experience. It was important to realize that mental disorders were often associated with chronic subdural collections, and such patients were frequently found in mental hospitals; but it was also important to realize that mental disorder might occur very early, as had been shown in Dr. Curtis's series.

PROFESSOR MAURICE EWING said that one of Dr. Curtis's patients was under his care at the Royal Melbourne Hospital. She had made a slow recovery, but now confabulated in a most delightful way and seemed to enjoy life. In future he himself would be more alert to detect those cases.

DR. GEOFFREY NEWMAN-MORRIS said that he was interested from the point of view of the "post-traumatic syndrome". He saw all too many of those cases on behalf of insurance companies, and he was convinced that they were not "compensation syndromes" but real entities; the patients had headaches and what he might call "snitchiness". He wondered whether they were suffering from subdural collections.

DR. KATE CAMPBELL said that in the infant cases quoted by Dr. Curtis the Wassermann test had not been carried out, but the majority of the mothers had not yielded positive Wassermann reactions before confinement.

DR. REGINALD HOOPER said that care was necessary in relation to the pathology. He had concluded that the pia-arachnoid in its intact state was practically impermeable to cerebro-spinal fluid, but if it was damaged by trauma or infection it became permeable, and in some cases, cerebro-spinal fluid came through the arachnoid like beads. He thought that thrombo-phlebitis was a classical cause of subdural collections in children, and if the protein content of the effusion was small it would gradually be absorbed. He considered that Dr. Curtis had done a great service in pointing out the value of early exploration in the cases under discussion, and he thought in all cases in which the condition became stationary, exploration should be carried out.

DR. JOHN ADEY said that it was well recognized in mental hospitals that acute confusion could occur after cerebral trauma, but if the patient survived, in many cases confusion gradually cleared up. Commenting on Dr. Newman-Morris's remarks on traumatic neurasthenia, Dr. Adey said there had been many cases in the last war, but not from war injuries; they usually occurred in dispatch riders after collisions. Those affected complained of headache and other symptoms, which were quite real, and when it was insisted that such patients should remain for three weeks in bed, after even minor head injuries, the results were greatly improved.

Dr. Curtis, in reply, said that most of the questions which had been raised had been answered. In one of his cases he considered that the subdural effusion was the result of staphylococcal infection, and trauma was not the only factor—infection played a role. He did not reject thrombo-phlebitis as a cause in such cases, but had not been able to prove it. He regarded the question of variation of incidence in different places as a thorny one; but there was little doubt that if cases were looked for, they would be found. With reference to the failure of the brain to expand, he considered that in some cases that was due to a further collection of fluid, but in the acute group it was, he thought, due to severe injury, of which fluid collection was only a part. The situation was comparable with that occurring in cases of acute subdural hæmorrhage, in which often, after early improvement, there was relapse because of underlying severe brain damage.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE MEDICAL BOARD IN SOUTH AUSTRALIA.[1]

[From the *Australasian Medical Gazette*, April, 1890.]

THOUGH upwards of three months have elapsed since the passage of the amending Medical Act in South Australia, no Medical Board has yet been appointed to take the place of the old one, the members of which so unselfishly resigned on August 26, 1889, from conscientious objections to the liability which, by a decision of the Supreme Court, they were under to register diplomas that they considered were of an untrustworthy character. As a consequence of there having been no Medical Board for seven months in that colony, no list of medical practitioners has been published for this year and no new arrivals have been registered, the medical superintendent and four other resident medical officers of the Adelaide Hospital being unregistered at the present time. These gentlemen are, of course, fully qualified, but the anomaly remains that the metropolitan hospital has five unregistered medical practitioners for its resident staff, which we are sure must be in contravention of the constitution under which it has been established. The continued

[1] From the original in the Mitchell Library, Sydney.

neglect of such a vital matter as the appointment of a Board for the carrying out of the Acts regulating the practice of medicine is not creditable to the Government of South Australia, and is less so when the Premier is a member of the profession.

Correspondence.

KALA-AZAR IN A CHILD OF FIVE YEARS.

SIR: Dr. Rigg's report on the child with Mediterranean kala-azar (March 23, 1957) has only recently come to my notice, and, as it raises some important issues, I would like to make a few comments which may be helpful to any of your readers who may encounter a similar problem.

Firstly, regarding the choice of drug. One of the extraordinary things about kala-azar is that the results of antimony treatment vary according to geographical factors. For example, a good response can be expected in the Indian and Chinese forms of the disease, but the Sudanese and Mediterranean forms respond poorly. This, however, is only a generalization, for "Solustibosan" (the Bayer product of sodium antimony gluconate) has been successfully used in the Sudan by Kirk and Sati,[1] and antimony tartrate, which proved a failure in China, has been used by Izzo[2] in Italy with good results. The situation has been summed up by Napier,[3] when he says that there is a drug of choice for each geographical area, and, within a given area, a patient who is resistant to one antimony compound will often respond to another.

When Kirk[4] reported promising results with pentamidine (an aromatic diamidine) in the treatment of kala-azar in the Sudan, it was hoped that it would prove successful in Mediterranean kala-azar, but this hope has not been fulfilled. Dr. Rigg's disappointing experience with this drug has been shared by many before him, and I agree with Dr. Dudley Hagger (April 27) that it is a pity antimony was not tried before resorting to splenectomy. I remember Sir Neil Hamilton Fairley's wise counsel against splenectomy when we had to treat a child of 19 months, desperately ill with Mediterranean kala-azar, in whom pentamidine had failed. A "normal" course of "Pentostam" (the Burroughs Wellcome preparation of sodium antimony gluconate) for a child weighing 11 kilograms (24 pounds) was equally unsuccessful, and we had to give a course of over twice the previous dosage before obtaining any improvement. This consisted of a total dosage of 0·22 gramme per kilogram of body weight instead of the normal 0·1 gramme per kilogram of body weight, divided into 15 daily intramuscular injections, the last seven injections being of 0·2 gramme (two millilitres) each. This course was repeated after an interval, and the child has made what appears to be a complete recovery. As Mediterranean kala-azar usually affects children, I suspect that a common cause of failure with antimony is that calculations of dosage according to body weight do not take into consideration the diluting effect of the large quantity of blood stored within the grossly enlarged spleen.

Secondly, regarding the dangers of antimony therapy. When Dr. Rigg says "It was decided . . . not to use any antimony preparation to treat the disease because of the considerable hazard of toxicity", I think he is being unduly pessimistic. When I treated large numbers of children for schistosomiasis in Southern Rhodesia using various trivalent antimonials, I was struck by the absence of serious toxic effects, and it is my impression that children tolerate antimony compounds better than do adults. Izzo[2] met virtually no side-effects using antimony tartrate in treating 240 children for kala-azar in Naples. So far as pentavalent antimonials are concerned, the toxic effects which have been recorded with the older preparations such as urea stibamine (Napier[5]) are extremely rare with sodium antimony gluconate (Schmidt[6]), and I have not observed any untoward reactions when this compound in the form of "Pentostam" has been used in this hospital.

Thirdly, regarding the future management of Dr. Rigg's patient. I think it should be appreciated that the removal of the spleen may not have eradicated the infection, even though four courses of pentamidine succeeded in freeing the sternal marrow of Leishman-Donovan bodies, for the infection may smoulder in the liver. Das and Sen Gupta[1] have shown that kala-azar may become active again after splenectomy, and, in view of this, it would be advisable to examine the child at regular intervals for several years to come.

Yours, etc.,

Hospital for Tropical Diseases,
4 St. Pancras Way,
London, N.W.1.
December 19, 1957.

W. H. JOPLING.

[1] *Ann. Trop. Med. & Parasitol.*, 1947, 41: 14.
[2] *Acta med. ital.*, 1957, 12: 128.
[3] *J. Trop. Med. & Hyg.*, 1949, 52: 244.
[4] *Tr. Roy. Soc. Trop. Med. & Hyg.*, 1939, 32: 533.
[5] *Indian M. Gaz.*, 1926, 61: 559.
[6] *J. Trop. Med. & Hyg.*, 1950, 53: 95.

"BE STRONG AND OF A GOOD COURAGE."

SIR: In the course of his Rupert Downes Memorial Lecture (M. J. AUSTRALIA, December 21, 1957, page 892), Major-General Sir Kingsley Norris made an enfilading attack upon the Australian Army Rifle Clubs. This attitude, which appears to become intermittently fashionable among some of the senior defence and administrative officers, is apparently based upon various misconceptions as to the functions and utility of the rifle clubs (the Minister for the Army, Mr. Cramer, when presenting the prizes at the Queen's Prize Shoot on Anzac Range, Sydney, on October 11, admitted that that was the first occasion he had been on an army rifle range).

The opinion seems to be that the rifle clubs are merely parasites upon the Defence Department, enjoying cheap sport at national expense — taking all and giving nothing in return. Apart from other essential non-service gear, which they buy elsewhere at market prices, rifle club members purchase their service rifles and associated parts from Ordnance very cheaply, and pay considerably more to have them put into a serviceable target-shooting condition. Since these rifles and parts are recoverable stores, they represent not a charge upon, but a net gain to, the army, equal to their nominal purchase price — which means that the army sells its cake and still has it in the long run too.

Ammunition is acquired from army, part free issue to, and part bought by, the clubs for cash, per efficient member. Since this ammunition, if not used by riflemen, would be disposed of by destruction, yielding a net return of nil, it cannot reasonably be claimed that we represent a charge upon them in this respect. A large quantity of ammunition was destroyed adjacent to the range during the last Queen's Shoot in Sydney, rather robbing of its point the Minister's complaint that we were costing his Department too much for ammunition.

These, however, are probably minor, albeit important, points. The main issues generally seem to boil down to: (*a*) the rifle clubs cost the country too much — the Commonwealth subsidy in the last Budget was £77,000, not the £600,000 erroneously quoted by General Norris; (*b*) that training in the use of the rifle is, or will be, unnecessary for the soldier of the future with his automatic weapons.

With regard to the first point, it may be of interest to the General to point out that most of the subsidy is spent on part of the cost of maintenance of rifle ranges throughout the Commonwealth, the rest, and greater part of the necessary expenditure in this direction, being found by the rifle clubs — £30 grant/£90 club expenditure was the ratio in this small club last year; and the ranges maintained by the clubs on this basis are used by local military formations for their regular small arms training and efficiency shoots, both rifle and automatic weapons. Hence the army has maintained for it, and available on demand, safe range areas throughout the Commonwealth for weapon training. Physically the rifle clubs maintain these wholly, and for the greater part financially, without retaining any rights on the property whatsoever.

As to the second point, which is a hotly debated one, I would make two submissions: whilst the ability to hit a small bullseye at 1000 yards (not a range used now, anyhow) might be considered "lily-painting" (and a darned sight harder and needing a lot more training to do), the military effectiveness of an automatic small arm can only be increased when used accurately by a man who learnt to use a rifle properly as a member of a rifle club, he being, besides, a more apt recruit to convert to the former weapon. In contradistinction is the recruit who, unversed in the use and techniques of accuracy with a rifle, looks upon the automatic weapon as a rather exotic form of shotgun wherein the volume of fire compensates for his lack of accuracy; quite apart from the problems of manufacture, distribution and supply of the enormously increased expendi-

[1] *Lancet*, 1950, 2: 681.

ture of ammunition so entailed. Further, since these automatic small arms have become essential to modern armies, you will have to have safe places available in which to train men in their use — where else than on the rifle ranges?

"I appeal to the young men in the community to take an active interest in the rifle club movement. People are saying without any support, however, that the day of the rifle is finished and that the next war will be fought by merely pushing buttons. That is totally untrue, and man is still the first weapon of war, whilst the first weapon of man is the rifle, then the tommy-gun, and so on. Believe me, the rifle will always be a big factor in winning or, in its absence, of losing battles. I therefore commend rifle shooting and the use of the rifle to everybody."—Field Marshal Viscount Montgomery.

I would respectfully suggest to General Norris that the army gets more out of the rifle clubs than it puts into them; and if he persists in thinking otherwise, then he does not know a good thing when he sees it.

Yours, etc.,

J. E. BINNIE, M.B., B.S.,
Honorary Secretary,
Kyogle Rifle Club.

Kyogle,
New South Wales,
December 21, 1957.

CARCINOMA OF THE LUNG: ALWAYS A SUSPECT.

SIR: Though this hospital is ceasing to function as a chest institution, a diagnostic and follow-up chest clinic with facilities for all necessary investigations is being maintained. No thoracic surgeon visits this hospital now and, therefore, when a diagnosis of a probable carcinoma is reached, or when surgery is indicated, the patients are referred to one of the major thoracic units in Sydney for admission. The following case we had recently might be worth recording because of the age of this patient.

M. (a male, aged 32) had a routine X-ray on September 17, 1957, which showed an area of consolidation in the left upper lobe. A repeat X-ray was carried out on November 12, 1957 (an earlier appointment had actually been given, but the patient was delayed because of family matters). This showed obvious increase in consolidation and the patient was referred to this clinic on November 18, 1957. He arrived about 9.30 a.m. and was able to leave at 3 p.m. In this interval a full clinical examination was carried out, and the following investigations were done: (i) follow-up postero-anterior X-ray; (ii) blood count and blood sedimentation rate; (iii) Mantoux test; (iv) sputum tests for acid-fast bacilli and secondary organisms; (v) left lateral tomography; and (vi) bronchoscopy (when necessary bronchography is done simultaneously). He gave a history of some blood-streaked sputum some weeks previously, otherwise he felt well and no abnormalities were detected on physical examination. In this particular case the findings were not conclusive; but carcinoma was still suspected on radiological appearances, and accordingly arrangements were made for the patient's admission to a thoracic unit in Sydney. There a left scalene node biopsy was performed, examination of which showed oat-cell carcinoma.

This case illustrates that the least possible delay existed here between pick-up, diagnosis and referral to surgeon, and yet the prognosis is unfavourable. The fact that this man is married and has five children (twins having been born to his wife 10 days previously) only adds further to the unfortunate circumstances of this case.

For some time now carcinoma of the lung has superseded tuberculosis in importance because of an increasing rate in case-finding and mortality. The estimated number of deaths (from carcinoma of the lung) in England and Wales for 1955 was 17,000. In the more forward countries tuberculosis is rapidly being brought under control, and some of these countries are now setting up centres for the early detection of malignant disease.

Due emphasis has often been placed on the early diagnosis of lung carcinoma, as only then is radical surgery with hope of recovery made possible. It has been stated that much of the delay between onset of symptoms and seeing the surgeon often lies with the chest physician. In his review, Vernon Thompson in 1957 found an average delay of 19·2 weeks from chest physician to operation. The comparative figure of E. N. Moyes (1957) was 6·6 weeks. With better organization these figures should be considerably decreased.

Whenever a carcinoma of the lung is suspected it has always been our policy to carry out all the necessary investigations with the least possible delay and surgical consultation held within a week of our first seeing the patient. Indeed, as the above case illustrates, the investigations could be completed on the same day. The patients are not alarmed at being put through the whole machinery. Once the problem is discussed with them, we have usually found them most cooperative and grateful for what has been done for them.

Even if after all the necessary investigations the diagnosis is only a tentative one, a surgical consultation should still be sought without delay, as so often a diagnostic thoracotomy is obviously indicated. This applies even more so today, because in the right hands the risks attached are negligible.

H. P. Longstreth *et alii* in 1957 published a series of cases in which a diagnostic thoracotomy was carried out. They had no mortality or morbidity and, moreover, they claim that in some cases it helped to institute appropriate treatment immediately, thus avoiding unnecessary prolonged treatment of such cases as "tuberculosis". They also found that the undiagnosed patient can be saved considerable inconvenience and his prognosis improved because of earlier surgical intervention. In their opinion, the practice of "watchful waiting" is no longer justified.

The facilities of chest clinics are at the disposal of all medical practitioners alike. All cases presenting with unexplained cough, wheezing or dyspnœa, recurring respiratory infections, small repeated hæmoptyses, should be fully investigated with the least possible delay to exclude carcinoma of the lung. The greatest possible cooperation between general practitioner, chest physician and thoracic surgeon would ensure an earlier diagnosis and, therefore, a better chance for radical treatment. Indeed, it would also be relevant to express the hope that a diagnosis of possible carcinoma of the lung should be the password into any thoracic unit. In the interest of the patient, no waiting list should be allowed to build up.

Yours, etc.,

N. A. SALIBA.

Bodington Red Cross Chest Hospital,
Wentworth Falls,
New South Wales.
December 13, 1957.

PLANTS, INCLUDING FUNGI, POISONOUS OR OTHERWISE INJURIOUS TO MAN IN AUSTRALIA, SERIES V.

SIR: Those of your readers who are interested in this subject will find Series V of the above in *The South Australian Naturalist*, Volume 31, Number 3, pages 36 to 39, and Number 4, pages 53 to 58. Series IV appeared in this Journal in 1943.

Yours, etc.,

J. B. CLELAND.

The University,
Adelaide,
December 18, 1957.

SPRUE.

SIR: Your number of October 12 has just reached me.

I was interested to see the article by Dr. R. A. Douglas on sprue as he has seen it in Australian aborigines. As he points out, it has previously been considered to be a disease of Europeans only. However, his findings show that this is not so, and similarly we have found a large number of cases here among the Indian population.[1]

Dr. Douglas states that his cases responded to folic acid, which removed all symptoms and apparently cured the patient. This also is what the South American authors have found. However, Dr. Douglas does not state what his criteria were for normality.

There is no doubt that in every case folic acid will cause reversion of megaloblastic change in the bone marrow and will cure the anæmia (at least temporarily), even if that anæmia be due to B_{12} deficiency. However, if one studies these cases closely, it will often be found that folic acid makes no change in the number or character of the stools which the patient passes, and also no change in the

[1] *Indian J. M. Sc.*, 1957, 2: 687.

fat excretion. We have not been able to influence the level of fat excretion in cases of sprue by giving folic acid, nor by giving B_{12}, and I would seriously question the statement that these cases are due to a deficiency disease. As far as our cases here are concerned, I am sure that the disease is not due to a deficiency, but that all the deficiencies, whether they be B_{12}, folic acid, iron or other substances, are secondary to some intestinal disorder.

It would be very interesting to know whether or not the cases among the Australian aborigines really are cured by folic acid treatment or whether it merely cures their anæmia. It may well be, of course, that there are regional differences in the disease in different parts of the world, and I am therefore writing this letter to urge Dr. Douglas, or anyone else who may have access to cases of this type, to study the response to folic acid in greater detail. It would be desirable to put the patients on a continuous daily fat balance, and watch the effect of the folic acid administration on the fat excretion and on the nature and character of the stools.

Yours, etc.,

S. J. BAKER, M.D.,
Lecturer in Medicine.

Christian Medical College Hospital,
Vellore,
India.
December 21, 1957.

ACUTE APPENDICITIS IN A CENTENARIAN.

SIR: It may interest you and your readers to know that a female patient, aged 100 years and four months, was successfully operated upon for a gangrenous appendix at Balmain Hospital on the third instant. She presented with signs of an acute abdomen and a painful inguinal swelling on the right side. We opened into a femoral hernia, which had very thickened walls and was filled with a purulent fluid. The sac was removed and closed, and a paramedian incision made and a gangrenous appendix found and removed. The wound was closed, a tube inserted through a stab wound and the patient made an uninterrupted recovery. She was discharged from hospital on Friday, December 13.

I think this case is of interest, as I can find no reference in the literature to any acute appendix in anyone approaching this age, and would be pleased to hear of any similar case.

Yours, etc.,

ARTHUR SHAPPERE.

175 Macquarie Street,
Sydney,
December 16, 1957.

IMMUNIZATION OF RH-NEGATIVE POST-MENOPAUSAL WOMEN BY RH-POSTIVE BLOOD TRANSFUSIONS.

SIR: Dr. Krieger, in her article on "Immunization of Rh-Negative Post-Menopausal Women by Rh-Positive Blood Transfusions" in your Journal of December 14, 1957, has performed a service by drawing attention to the fact that even the most careful cross-matching is not infallible, and that every transfusion must therefore be recognized as a calculated risk.

None the less it must be stressed that her findings should not destroy confidence in the value of cross-typing. It is not a futile procedure, but is as safe as any biological test can be, and moreover affords the only measure of protection available to those receiving transfusions, or for that matter to those performing them.

It is well recognized by the blood transfusion services that the giving of homologous blood is desirable in the great majority of cases, and the fact that Rh-positive blood may be issued for Rh-negative patients who show no evidence of previous sensitization, and who will not in future bear children, means simply that this is the best that can be done for those particular patients under existing circumstances of supply. The choice lies between Rh-positive blood and none at all. Certainly all the Rh-negative blood which is available is used up on patients who could not receive any other type. Were there sufficient Rh-negative donors on the panel, homologous blood would undoubtedly be issued for all Rh-negative patients.

The evidence that any serious harm can arise from the transfusion of Rh-positive blood to Rh-negative patients who show no indication of previous sensitization is far from convincing. In the cases where antibodies develop, the survival of the transfused cells may well be diminished, but this does not appear to be accompanied by any severe reaction. Even in the case where latent sensitization facilitates the rapid production of antibodies, as postulated in Dr. Krieger's third case, the occurrence of severe reaction must be so rare as to fall within the range of experimental fallibility. It is, however, a pity that we were not aware of the patient's previous history at the time the blood was requested for her.

The problem would be solved if more doctors and hospitals using our service took an interest in obtaining new donors from among the relatives of their patients. With the full cooperation of the profession in this regard, many of these worrying factors might be avoided.

Yours, etc.,

J. P. MORRIS,
Director, Australian Red Cross Society (Victorian Division) Blood Transfusion Service.

Royal Melbourne Hospital,
Melbourne,
December 19, 1957.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Annual Subscription Course.

THE Post-Graduate Committee in Medicine in the University of Sydney announces the following lectures in a course arranged around the visit to Sydney of Professor H. Olivecrona, Serafimeriasarettet, Stockholm: Friday, January 31: 1.30 p.m., Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, seminar, "Cerebellar Spongioblastomas". Thursday, February 6: 8.15 p.m., Stawell Hall, 145 Macquarie Street, "Hypophysectomy in the Treatment of Malignant Disease". Monday, February 10: 8.15 p.m., Stawell Hall, 145 Macquarie Street, "Trigeminal Neuralgia". Wednesday, February 12: 2 p.m., Maitland Lecture Theatre, Sydney Hospital, "Treatment of Acoustic Tumours".

Professor Olivecrona will also visit the following centres: Friday, January 31, 11 a.m., Royal Prince Alfred Hospital: Neurosurgical department. Monday, February 3, 2 p.m., St. Vincent's Hospital: Clinical discussion. Wednesday, February 5, 2.30 p.m., Royal Prince Alfred Hospital: Neurological consultative clinic. Thursday, February 6, 11 a.m., The Royal North Shore Hospital: Presentation of cases. Monday, February 10, 2.15 p.m., Royal Alexandra Hospital for Children: Case demonstration and discussion. Tuesday, February 11, 11 a.m., Brain Research Unit, Physiology Department, the University of Sydney.

The above programme will be available to all members of the annual subscription course.

General Practitioner Courses, March, 1958.

The Post-Graduate Committee in Medicine in the University of Sydney announces that the following week-end courses for general practitioners will be held in Sydney during March, 1958.

A course in neurology will be held at The Royal North Shore Hospital of Sydney on Saturday, March 15, from 2 p.m. to 5.15 p.m., and on Sunday, March 16, from 9.45 a.m. to 5.15 p.m. The fee for attendance is £3 3s., and programmes are now available.

A course in electrocardiography will be held at Sydney Hospital on Saturday, March 22, from 10 a.m. to 5 p.m., and on Sunday, March 23, from 10 a.m. to 1 p.m. The fee for attendance is £3 3s., and programmes are now available.

Diploma Courses, March, 1958.

The following diploma courses will begin in Sydney in March, 1958.

A course of instruction for candidates for the final examination for Fellowship of the Royal Australasian College of Surgeons has been organized by the New South Wales State Committee of the Royal Australasian College of Surgeons in conjunction with the Post-Graduate Committee in Medicine. The course will be held from March 17 to April 25, full time, under the direction of the Professor of Surgery in the University of Sydney, Professor John

Loewenthal. The headquarters for candidates will be the Harold Dew Room at the New Medical School. Lectures, demonstrations and clinical teaching will be conducted at each of the four teaching hospitals. Candidates must have passed the primary examination of one of the Royal Colleges. The course is limited, and early application to the Post-Graduate Committee in Medicine is essential. The fee for attendance is 20 guineas.

A course for Part I of the Diploma in Diagnostic Radiology of the University of Sydney will begin on March 17 and continue for a period of four months. The fee for attendance is 45 guineas, and copies of the by-laws and regulations governing this diploma may be obtained on application to the Committee.

A course for Part I of the Diploma in Therapeutic Radiology of the University of Sydney will begin on March 17 and continue for a period of four months. The fee for attendance is 45 guineas, and copies of the by-laws and regulations governing this diploma may be obtained on application to the Committee.

Method of Enrolment.

Early written application for attendance at these courses should be addressed to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney."

General Revision Course, 1958.

The Post-Graduate Committee in Medicine in the University of Sydney announces that its annual general revision course will be held for two weeks from Monday to Friday, May 5 to 16, 1958, inclusive. The main theme of the course is therapeutics. As in former years, the programme will be a comprehensive survey of modern trends in diagnosis and treatment of special value to general practitioners, consisting of lectures, special demonstrations, symposia, discussions and conferences. Social activities will include a cocktail and buffet party, and the eleventh Annual Post-Graduate Oration in the Great Hall of the University of Sydney, to which all members of the course and their wives will be invited. A golf competition for the Post-Graduate Golf Cup, open to members, will also be held, and the Brydon Cup will be for competition by country members. The fee for attendance is £12 12s., full time, or £6 6s. for one week or part time. Fees and travelling expenses incurred by practising members of the profession in attending this course are allowable income tax deductions.

Early written application, enclosing remittance, should be sent to the Course Secretary, The Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Telephones: BU 4497-8. Telegraphic address: "Postgrad Sydney."

FEDERATION OF COUNTRY LOCAL ASSOCIATIONS (NEW SOUTH WALES).

Post-Graduate Week at Canberra.

THE seventh annual post-graduate week of the Federation of Country Local Associations will be held at Canberra from April 14 to 18, 1958. In addition to an interesting scientific programme, highlights of this course include the following: a visit to the John Curtin School of Medical Research, where advanced research techniques will be demonstrated; a conducted tour of the Research School of Physical Sciences; a trip to Mt. Stromlo, where the new telescope will be demonstrated; a visit to the Commonwealth Scientific and Industrial Research Organization, etc. Further particulars may be obtained from the Course Secretary, Dr. A. G. Cumpston, Post Office Box 365, Canberra City, A.C.T.

CLINICO-PATHOLOGICAL CONFERENCES AT SYDNEY HOSPITAL.

THE Director of the Clinical Research Department, Sydney Hospital, asks us to announce that clinico-pathological conferences at the hospital will be held as usual during 1958. The meetings will take place in the Maitland Lecture Theatre at 5 p.m. on the third Tuesday of each month, from January 21 to November 18.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED DECEMBER 21, 1957.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	2(2)	7(1)	..	..	..	..	..	9
Amoebiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	2	..	..	..	..	..	2
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhoea (Infantile)	1(1)	14(14)	9(1)	..	..	..	..	..	24
Diphtheria	3(1)	..	..	..	..	..	..	..	3
Dysentery (Bacillary)	..	..	2(2)	6(6)	2(2)	..	4	..	14
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	1	..	..	..	..	..	..	..	1
Infective Hepatitis	25(13)	17(9)	5	2(2)	5(1)	1	1	..	59
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	..	..	..
Leptospirosis	..	..	..	..	..	..	..	..	..
Malaria	..	..	1	..	..	..	..	..	1
Meningococcal Infection	2(2)	..	2(1)	..	..	..	..	..	4
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	..	..	..	..	..	..	..	..	..
Puerperal Fever	1	..	2	..	..	..	..	..	3
Rubella	..	56(43)	5(5)	10(6)	41(34)	..	..	..	112
Salmonella Infection	..	..	..	..	1(1)	..	1	..	2
Scarlet Fever	11(9)	19(13)	2(1)	4(3)	3(3)	1(1)	..	..	40
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	2	..	..	..	..	..	2
Trachoma	..	..	..	..	4	..	..	..	4
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	18(13)	15(11)	8(5)	6(6)	8(4)	4(3)	1	..	60
Typhoid Fever	..	1(1)	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	2	..	..	..	..	..	2
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

The College of General Practitioners.

PROPOSED SOUTH AUSTRALIA FACULTY.

A MEETING of the College of General Practitioners is to be held at Newland House, 80 Brougham Place, North Adelaide, on Saturday, February 8, 1958, at 8 p.m. All medical practitioners who are interested in the proposal to establish the South Australia Faculty of the College are invited to attend this meeting.

Congresses.

FOURTH INTERNATIONAL GOITRE CONFERENCE.

THE American Goiter Association and the London Thyroid Club announce that the fourth International Goitre Conference will take place in London from July 5 to 9, 1960. This will precede the International Endocrine Conference in Copenhagen by ten days. Scientific sessions will be held under the presidency of Sir Charles Harington at the Royal College of Surgeons, Lincoln's Inn Fields, on July 6, 7 and 8. Participants will be required to pay a registration fee of £4 sterling or $10 U.S. Proceedings will be published in English, French, Spanish and German. Those who desire to submit papers for consideration by the Programme Committee should send abstracts not later than December 31, 1959, to either of the following: Dr. Selwyn Taylor, 3 Roedean Crescent, London, S.W.15; Dr. John C. McClintock, 149½ Washington Avenue, Albany 10, N.Y., U.S.A. A very few honoraria will be available for travelling expenses; particulars may be obtained from Dr. Selwyn Taylor or Dr. J. C. McClintock. Hotel reservations may be secured from Thomas Cook and Son, Limited, and it is expected that special travel facilities will be offered by B.O.A.C.

Naval, Military and Air Force.

APPOINTMENTS.

THE following appointments, changes etc. are promulgated in the *Commonwealth of Australia Gazette*, No. 1, of January 2, 1958.

ROYAL AUSTRALIAN AIR FORCE.

Permanent Air Force.

Medical Branch.

The following Flight Lieutenants are promoted to the rank of Squadron Leader: D. M. Newman (013992), A. Cameron (039928) (Acting Squadron Leader), W. Blake (014736), E. H. Stephenson (0312182) (Acting Squadron Leader), R. P. V. Checcucci (0311661) (Acting Squadron Leader).

Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Bagdonavicius, Irene, M.D., 1949 (Univ. Tübingin) (provisionally registered in accordance with the provisions of Section 17 (1c) of the *Medical Practitioners Act*, 1948-1957), 124 Amy Street, Regent's Park, New South Wales.

Eikens, D. Arnolds, M.B., 1955 (Univ. Riga) (provisionally registered in accordance with the provisions of Section 17 (1c) of the *Medical Practitioners Act*, 1938-1957), 41 Townsend Street, Bankstown, New South Wales.

Deaths.

THE following deaths have been announced:

FREWIN.—Oscar Westcott Frewin, on January 1, 1958, at Adelaide.

WALKER.—Allan Seymour Walker, on January 8, 1958, at Sydney.

ROBINSON.—Arthur Thomas Rowlandson Robinson, on January 9, 1958, at Sydney.

Diary for the Month.

JAN. 21.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JAN. 22.—Victorian Branch, B.M.A.: Council Meeting.
JAN. 24.—Queensland Branch, B.M.A.: Council Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

Queensland Branch (Honorary Secretary, 88 L'Estrange Terrace, Kelvin Grove, Brisbane, W.1): All applicants for Queensland State Government Insurance Office positions are advised to communicate with the Honorary Secretary of the Branch before accepting posts.

South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide): All contract practice appointments in South Australia.

Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2-3.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this Journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

SUBSCRIPTION RATES.—Medical students and others not receiving THE MEDICAL JOURNAL OF AUSTRALIA in virtue of membership of the Branches of the British Medical Association in Australia can become subscribers to the Journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rate is £5 *per annum* within Australia and the British Commonwealth of Nations, and £6 *per annum* within America and foreign countries, payable in advance.